DECEMBER, 1950

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Emergency Gastrectomy in the Surgical Treatment of Perforated Peptic Ulcer

The Early Diagnosis of Carcinoma of the Cardia of the Stomach

The Management of Pancreatitis

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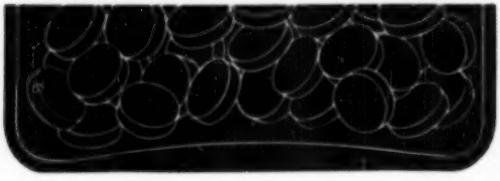

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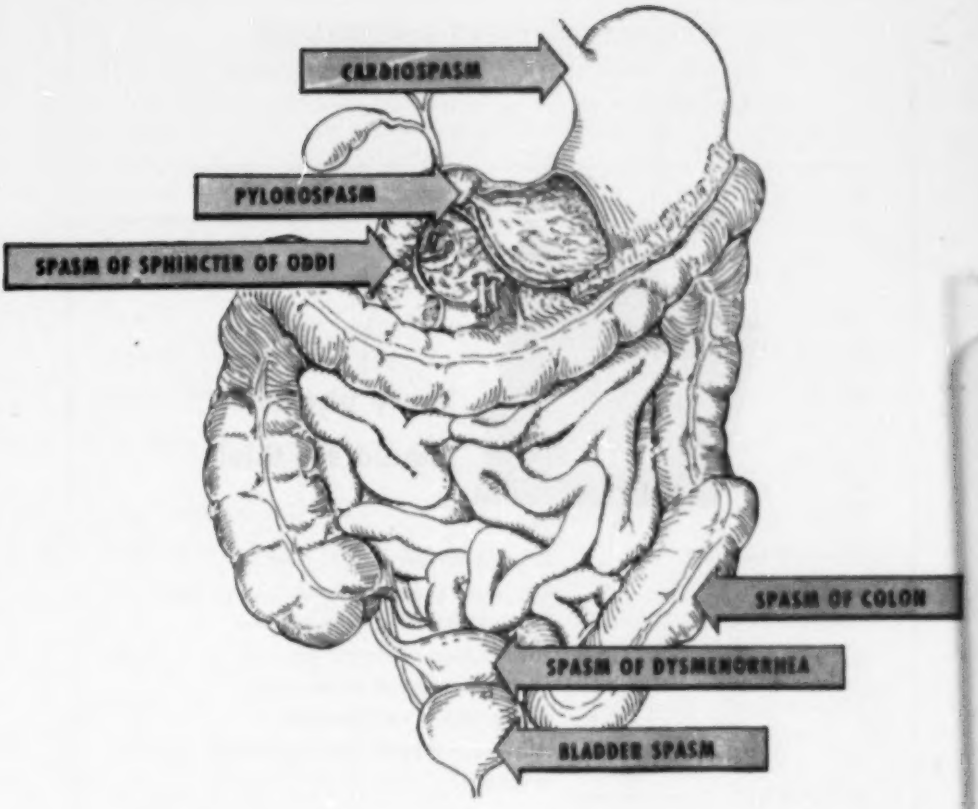

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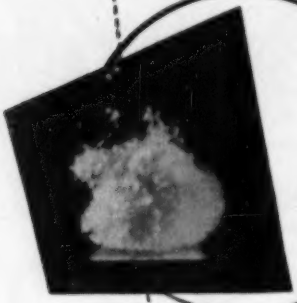

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(Incorporating the American Journal of Gastroenterology)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

Volume 17 DECEMBER, 1950 Number 12

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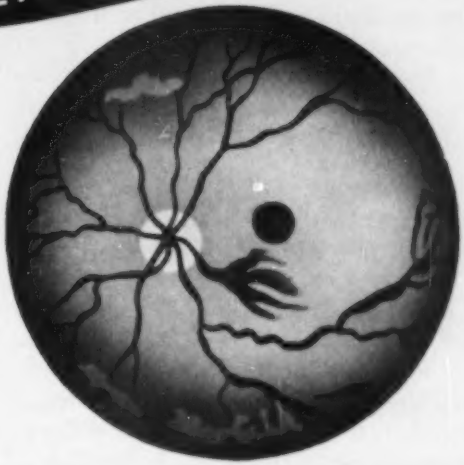

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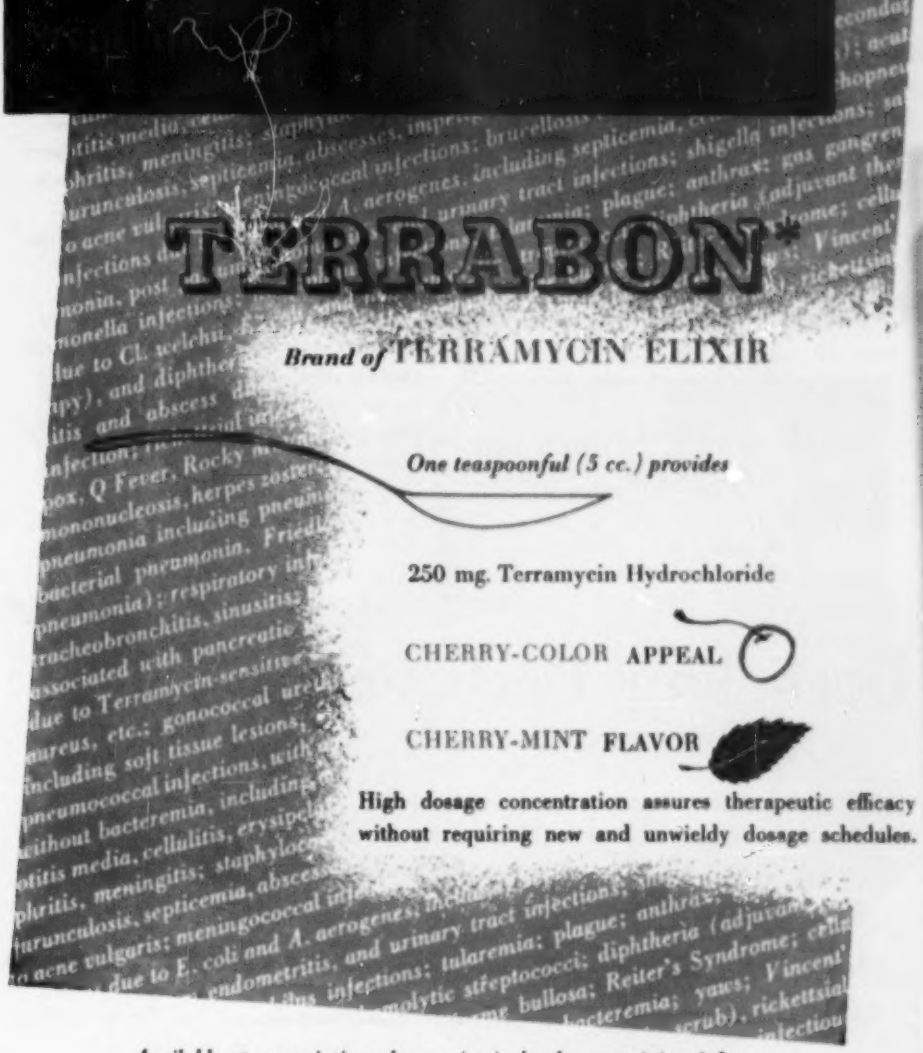
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Volume 17 DECEMBER, 1950 Number 12

THE EARLY DIAGNOSIS OF CARCINOMA OF THE CARDIA OF THE STOMACH *

MAX DANNENBERG, M.D.
and
IRVING K. ETTMAN, M.D.
Brooklyn, N. Y.

The importance of early diagnosis of carcinoma of the cardiac end of the stomach has become more apparent now that the surgical treatment of carcinoma of the fundus of the stomach has made such strides in recent years. It was

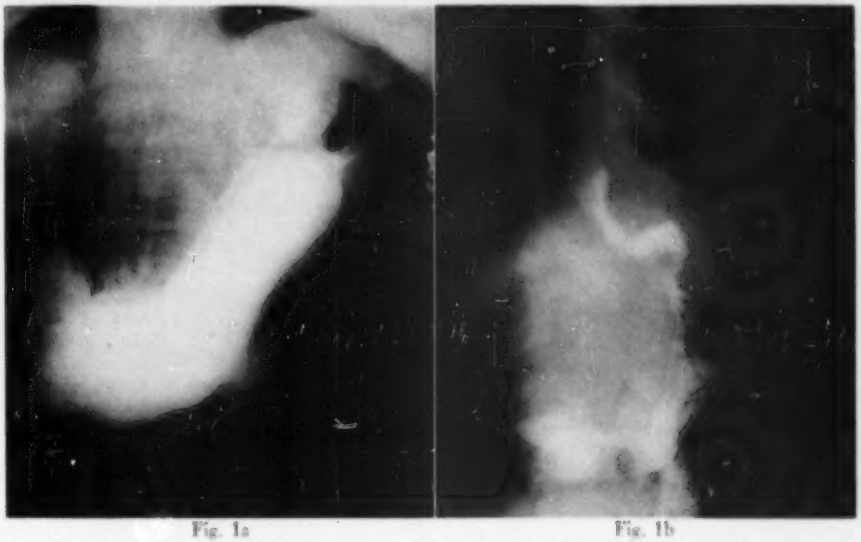

Fig. 1a Fig. 1b

Fig. 1—M.F. (a) Erect. Soft tissue mass density arising from the mesial and superior aspects of the cardia encroaching into the gas bubble.

(b) Air contrast. Showing the mass density encroaching into the lumen of the cardiac end of the stomach. Carcinoma confirmed by operation. Total gastrectomy.

Phemister[5] and his associates in 1938 who performed the first successful transthoracic resection for carcinoma of the lower end of the esophagus invading the cardiac end of the stomach. Sweet[6] has pointed out that the distressful symptom of dysphagia is eliminated by resection and primary anastomosis within the thorax.

*From the Department of Roentgenology, Beth-El Hospital, Brooklyn, N. Y.

Roentgenology has been an invaluable aid in presenting early cases of carcinoma of the cardia for surgery. Not infrequently, it is the only method of diagnosing this lesion. Gastroscopy is not always possible and is often unreliable. Gastric analysis cannot be relied upon and a hyperchlorhydria does not exclude the possibility of carcinoma of the fundus of the stomach.

In the past few years, we have noticed that cases of carcinoma involving the cardia of the stomach were recognized more often. The number of people who were aided by surgery was small. At operation, the tumor involvement was extensive and a large percentage of the patients had widespread metastases to the liver and lymph nodes. Many of the cases were previously x-rayed for gastric pathology and no evidence of a lesion could be demonstrated. These patients

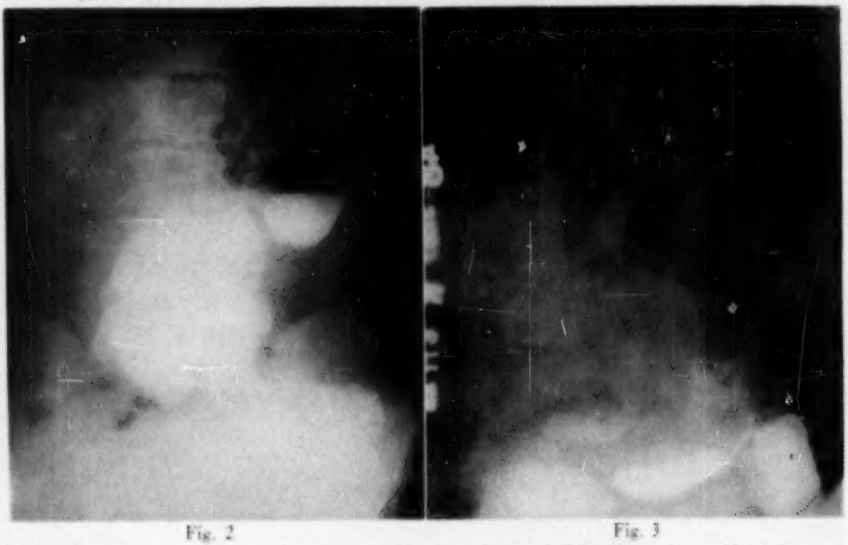

Fig. 2 Fig. 3

Fig. 2—M.Z. Large ovoid mass density protruding into the gas bubble. There is a wide contracture on the greater curvature aspect below the site of the lesion. Malignant neoplasm, confirmed by operation.

Fig. 3—M.F. Lesion of cardiac end suspected on routine study. The gas bubble was overdistended, revealing a soft tissue mass density. Proven by operation.

would return for re-examination because of the persistence of symptoms. On re-examination, an inoperable lesion of the cardia was found.

In a review of the histories and roentgenograms of one thousand gastrointestinal studies that were referred to the x-ray department to rule out gastrointestinal pathology, twenty-five cases of carcinoma of the cardia of the stomach were found. There were eighteen males and seven females. The ages varied from 47 years to 77 in our series. The age incidence was 47 plus.

Clinical History

A complete clinical history is of paramount importance in the correlation of the findings in lesions of the cardia. However, the absence of symptoms refer-

able to the gastrointestinal tract does not rule out the presence of a lesion. We have found this to be so in several of our cases.

The following symptoms were most frequently present:

1. Loss of appetite which often began with a distaste for a particular kind of food.
2. Difficulty in swallowing, especially solid foods.
3. Nausea and vomiting.
4. Weakness and anemia.
5. Hematemesis or tarry stools or both.
6. Regurgitation of food.
7. Loss of weight.
8. Epigastric pain.
9. Substernal pain.

Fig. 4

Fig. 5

Fig. 4—I.O. Tumor mass encroaching into the gas bubble, best visualized in the erect position, confirmed by operation.

Fig. 5—L.B. Irregular soft tissue mass density arising from lesser curvature in the cardia, encroaching into the gas bubble. Patient refused operation.

Roentgen Findings

For the purpose of the radiographic examination, the stomach may be considered anatomically in three parts[2]: the cardia or fundus, the body or media, and the antrum. The cardia is that portion which is above the level of the cardiac orifice of the esophagus; the body passes downward, from the level of the cardiac orifice, along the spine, to the line where the stomach turns across the vertebral column at the angle incisura. The antrum is that portion of the stomach from the angle incisura to the pylorus.

The surface of the mucous membrane is thrown up into folds, the rugae. As a rule, the rugae lying near the lesser curvature in the pars media and the antrum run parallel with the long axis of the stomach, while those in the fundus radiate in all directions. The mucosal contours around the cardiac orifice of the esophagus may produce a stellate appearance[4]. The star that is produced by the protrusion of the esophageal mucosa into the cardia is usually demonstrated in the right anterior oblique prone position.

A lesion of the pars cardia which has not invaded the lower esophagus is very easily overlooked on the x-ray examination, involving especially the anterior

Fig. 6—L.T. History suggested a lesion in cardiac end of stomach. The tumor mass was only demonstrated after the gas bubble was exaggerated with a Seidlitz powder. Operation confirmed malignant mass in the cardia.

and posterior aspects. With this fact in mind, we pay particular attention to the cardiac portion during the roentgenoscopic and roentgenographic examination.

The air bubble or magenblase plays an important role in diagnosing a lesion of the cardia. Here we make use of the air present in the stomach as a contrasting media. The normal bubble is pear shaped, and any distortion or encroachment will become easily apparent. Kirklin and Gilbertson[6] were able to diagnose tumors of the cardiac portion of the stomach by routine roentgenograms of the chest by noting encroachment or obscuration of the air bubble without the use of the opaque meal.

A procedure that is easily performed is the exaggeration of the gas bubble by means of an effervescent powder, after first coating the gastric mucosa with a barium mixture. In several cases where the symptoms and clinical findings were highly suggestive of a lesion in the cardia and routine roentgenoscopic and roentgenographic examination did not reveal the lesion, the air and barium contrast method were used. Thus, this method frequently demonstrated a lesion which was invading the cardia. This procedure is a simple one. The patient drinks a wine glassful of a thin barium mixture and is then placed in an upright position, in the prone and supine positions to coat the gastric mucosa. The patient drinks a fresh mixture of Seidlitz powder in water and is x-rayed in the erect position. Roentgenograms are then taken in the posteroanterior, right and left posterior

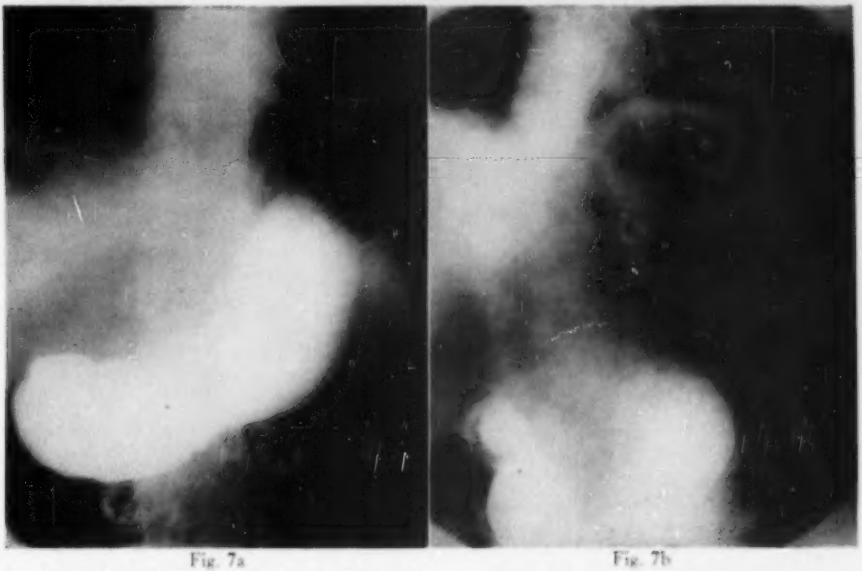

Fig. 7a Fig. 7b

Fig. 7—M.D. (a) Prone position: No definite defect demonstrable.
M.L. (b) Erect shows a mass density in the cardia mesially encroaching into the gas distended lumen.

oblique views. Any neoplasm that may be present will readily lend itself to view, and appear as a soft tissue mass encroaching into the air bubble.

The method has been described before but very often neglected. It is simple to perform, and gives us valuable information. There is no danger attached to this method as compared with the Levine tube[9] in distending the gas bubble. In one case, in spite of the usual precautions taken while passing the Levine tube, a rather profuse hemorrhage was started in a fungating neoplasm involving the cardia.

Lesions of the cardia of the stomach can often be detected on fluoroscopy with the patient in an upright position and rotated in the right anterior oblique

position. As the barium passes through the esophagus and enters the stomach, there may be a diversion or splitting of the stream.

Compression studies of the fundal region of the stomach are of little value, as pointed out by Bockus[1], since the rugal folds in this region are normally so coarse that one must be cautious in ascribing slight constant irregularities to malignancy. Kirklin[2] has pointed out that examination in the Trendelenburg position is not of great importance, since the fundus is filled by the opaque meal and a lesion in the cardia will be covered and obscured. It is also important in examining the stomach to avoid overdistention with the barium mixture, as this will obscure any lesion. The value of giving small amounts of opaque media cannot be urged too strongly.

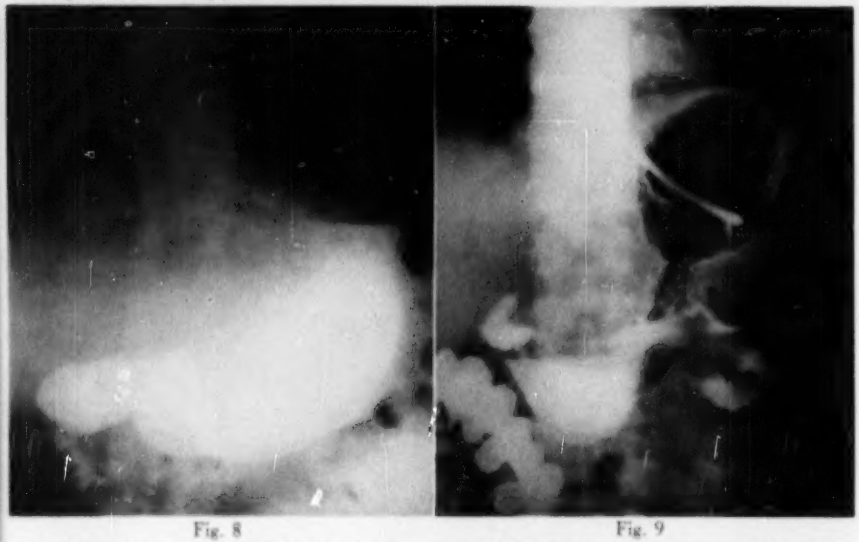

Fig. 8 Fig. 9

Fig. 8—R.S. An irregular nodular soft tissue mass arising from the lesser curvature, the cardiac end, encroaching into the gas bubble.

Fig. 9—Gas bubble exaggerated with air passed through Levine tube. There is a soft tissue mass on the posterior wall of the fundus.

Interpretation of Findings

1. A soft tissue mass protrudes into the lumen of the air-filled cardia.

2. An infiltrating tumor may destroy the mucous membrane. The absence of the mucosal folds as they approach the fundus is very suggestive of mucosal destruction.

3. Irregularities in the barium shadow as it passes down the esophagus indicated infiltration of the lower end of the esophagus.

4. In the fully distended fundus of the normal stomach, the smooth margin follows the contour of the diaphragm. The space between the diaphragm and the fundus may be invaded by a fungating mass, and the distance increased. How-

ever, one must be certain that the fundus of the stomach is fully distended, for a partially distended stomach will give the same finding[7].

5. The stellate appearance produced by the protrusion of the esophageal mucosa in the cardia must not be mistaken for a malignancy. This star shaped appearance is demonstrated in the prone position while tumors are seen at all angles. In a tumor, the barium will pass around the defect instead of through it.

6. A high left diaphragm will often put us on guard to rule out pathology of the cardiac end of the stomach.

Differential Diagnosis

The encroachment of the gas shadow by a tumor must be differentiated from an enlargement of the heart, ribs, spine, liver, and retroperitoneal masses. Recently we encountered a case where an accessory spleen, which was bound down by adhesions, produced a persistent defect in the cardiac portion of the stomach. The diagnosis was made on operation when the accessory spleen was found.

Conclusion

The clinical history is an invaluable aid in the early detection of the carcinoma involving the cardia of the stomach. A simple method of exaggerating the gas shadow by effervescent powder will often reveal a lesion in the cardia that is overlooked on examination in the routine methods which are commonly employed.

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THE MANAGEMENT OF PANCREATITIS*

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In considering the principles underlying the treatment of inflammatory disease of the pancreas, it is most convenient to consider acute and chronic pancreatitis separately as the two main entities. However, it is important to bear in mind that in some instances both phases may coexist. In cases now designated as chronic relapsing or recurrent pancreatitis, the acute process may merely represent an episodic exacerbation in the course of a basically chronic disease.

Acute Pancreatitis

It is now recognized that there are two principal forms of acute pancreatitis, acute edematous or interstitial and acute hemorrhagic or necrotic. While these two forms differ considerably from the standpoints of severity and prognosis, both appear to be cut from the same cloth. Acute interstitial or edematous pancreatitis probably represents simply a milder degree of acute inflammation. Under certain circumstances this may progress to the more severe hemorrhagic or necrotic form, or the latter may be present from the outset.

In all cases of acute pancreatitis, the initial regimen should be conservative. Absolute bed rest is prescribed and food is withheld. The patient must be very closely observed from the outset. Blood pressure, temperature, pulse, and respiratory rates should be determined and recorded every 2 hours. The abdomen should be examined at least every 2 hours in order to follow changes in tenderness, rigidity and the character of any palpable abdominal mass. When the patient is initially seen, a series of blood chemical determinations should be made which ought to include those for sugar, urea nitrogen, chloride, carbon dioxide combining power, amylase, lipase, bilirubin, calcium, total protein, albumin and globulin. In addition, a complete blood count, hematocrit reading and electrocardiogram are also advisable. The sugar, calcium, amylase and lipase values should be followed serially with repeated determinations approximately every 24 hours. The white blood cell count, differential analysis and hematocrit should also be determined serially, the former at intervals of at least 12 hours, in order to gain information regarding the course of the inflammatory process and the degree of hemoconcentration. If there has been pronounced vomiting, and especially if there is chemical evidence of hypochloremic alkalosis, serum potassium concentration should also be determined.

To relieve pain, demerol would appear to be the opiate of choice. This may be administered in a dose of 100 mg. subcutaneously every four hours or oftener if necessary. Morphine sulphate is not looked on with favor because of the suspicion that it exerts a vagotonic action. If the patient is not in pronounced shock with marked hypotension, a pearl of amyl nitrite may be inhaled or nitroglycerin given

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sublingually in a dose of 0.6 mg. (1/100 gr.) in the hope of reducing duodenal and ductal spasm. Under the same circumstances, unilateral or bilateral paravertebral procaine block or tetraethylammonium chloride may be employed. If paravertebral block is done and is without untoward reaction but is only partially effective, the block may be repeated after 24 hours. If tetraethylammonium is employed the initial dose is usually 1.0 c.c. (equivalent to 0.1 gm. of tetraethylammonium chloride) intravenously. Should there be no untoward reaction to this dose, a larger dose may then be given (up to 5.0 c.c.), the amount depending upon the severity of pain and the reaction of the patient. The same or smaller doses may be repeated as frequently as every 4 to 6 hours if necessary until pain is relieved.

To combat shock, normal saline, plasma and blood are given intravenously. Glucose solutions, however, should be given only after the blood sugar level has been determined because hyperglycemia is frequently present. After the severity of the pain has been reduced, phenobarbital sodium, 0.12 gm. (2 gr.), may be given subcutaneously to reduce nervous excitation. This may be repeated in the same dosage every 6 hours as necessary.

To reduce pancreatic secretory activity, a nasogastric tube is inserted and the gastric contents removed by constant suction. This is done to prevent hydrochloric acid from entering the duodenum and provoking secretin stimulation of the pancreas. In order further to obtund the secretin mechanism, the administration of an antacid such as aluminum hydroxide gel, 8 c.c. (2 drams), is also wise unless achlorhydria is known to exist. Atropine sulfate, 0.6 mg. (1/100 gr.), is given subcutaneously and repeated in doses of 0.4 mg. (1/150 gr.) every 6 hours or earlier in the hope of both reducing pancreatic secretion and allaying spasm. Ephedrine sulfate, 50 mg. (¾ gr.), may also be given subcutaneously and repeated in doses of 25 mg. (⅜ gr.), every 6 hours or earlier with the idea of additionally reducing pancreatic secretion. Evidence has recently been advanced indicating that soy bean extract containing trypsin inhibitor may successfully combat the increase in circulating trypsin believed to be present in acute pancreatitis. The role of this preparation in clinical practice, however, has yet to be evaluated.

To combat infection and peritonitis, antibiotics are employed. It is my practice as a rule to give crystalline penicillin in aqueous solution in a dose of 100,000 units intramuscularly every 2 hours and dihydrostreptomycin in a dose of 0.5 gm. intramuscularly every 12 hours. If peritonitis is especially severe and extensive, I prefer to increase the dose of penicillin to as much as 200,000 units and occasionally even more, and the frequency of administration of dihydrostreptomycin to 0.5 gm. every 6 hours. Aureomycin may be given intravenously as a substitute for these antibiotics if desired.

To correct chemical derangements, insulin is given in amounts as required to control hyperglycemia and glycosuria. One must be careful, however, to avoid hypoglycemia since this is vagotonic and capable of provoking pancreatic secretion. If hypocalcemia should develop, calcium gluconate, 20 c.c. of 10 per cent solution or 10 c.c. of 20 per cent solution, may be given intravenously and repeated as required.

If, despite the therapeutic measures employed, acute manifestations grow more marked and there are signs of progressive peritonitis, severe hemorrhage or pancreatic suppuration, or if obstructive jaundice becomes increasingly more severe, surgical intervention may become necessary. The decision for operation, however, must be individually determined in each case. The surgical procedures employed should be the least extensive ones feasible under the circumstances. As a rule, these procedures entail drainage of the lesser peritoneal sac and/or external drainage of the biliary tract by means of cholecystostomy or by means of choledochostomy if the gallbladder has been previously removed.

On the other hand, if clinical improvement becomes evident with subsidence of inflammation, operation is indefinitely deferred. Under such circumstances, the patient is maintained at bed rest until all signs of inflammation have subsided. Food is given cautiously beginning with warm water and tea in 30 c.c. (1 oz.) amounts every hour and progressing gradually to albumin water, cereal gruels, strained fruit juices, clear, non-fatty, diluted broths, jello, junket, and eventually to a low fat-high protein-high carbohydrate diet. Hard candies and skim milk powder, 120 gms. in skim milk, 1,000 c.c., are useful means of supplying carbohydrate and protein.

Following complete subsidence, interval operations may be undertaken to correct associated biliary tract disease, pancreatic cyst or fluid collection in the peritoneal sac, if these are found to exist.

Chronic Pancreatitis

Conservative medical management should be given a thorough trial in cases of chronic pancreatitis before resorting to surgical procedures. The patient may be permitted to be ambulatory with daily nonvigorous exercise of which the best is simple walking. However, physical exertion should be carried only to the point of the beginning of tiredness. A diet which is high in calories (3,000), high in protein (150 gm.), high in carbohydrate (450 gm.) and relatively low in fat (70 gm.) is supplied. These values are proportionately reduced in those with diabetes mellitus. Approximately half the protein may be supplied in hydrolyzed form because of the deficiency in trypsin secretion which is commonly present. The only fats permitted are the natural ones present in milk, cheese and eggs. Alcohol should be strictly forbidden.

To relieve pain, the same drugs are used as in acute pancreatitis.

To combat deficiency of external pancreatic secretion, particularly as manifested by steatorrhea and creatorrhea, triple strength pancreatin (enteric coated tablets) in a dose of 5 gm. (75 gr.) after each meal is highly useful. The size of this dose is to be emphasized. It may be increased further or it may be diminished depending upon the degree of pancreatic insufficiency. To further aid fat absorption, polyoxethylene sorbitan monooleate (Tween 80), 5 to 10 c.c. (1-2 teaspoonsful) of solution or 3 to 6 capsules, 0.5 gm. (7½ gr.) each, may be given 3 times daily with meals. If there is much fat loss, it is also wise to administer calcium. This may be supplied as calcium gluconate in a dose of 2 gm. (30 gr.) 3 times daily by mouth.

For nutritional supplementation, Vitamin A, 25,000 units, twice daily, Vitamin D, 1,000 units, twice daily, and Brewer's yeast powder, 5 gm. (75 gr.), 3 times daily, may all be given by mouth. In addition, crude liver extract, (2 units per c.c.), may be given intramuscularly in a dose of 3 c.c. twice weekly.

For lipotropic action, choline base, 2 gm. (30 gr.) may be employed 3 times daily by mouth.

For hypochromic anemia which may be coexistent, ferrous sulfate, 0.2 gm. (3 gr.), may be prescribed 3 times daily.

For diabetes mellitus which may be present, insulin is given in amounts as required for control.

If *pain* is prominent and cannot be relieved by the medications suggested, paravertebral block may be attempted. If successful, but only temporary in effect, the block may be repeated as required.

Surgical treatment of chronic pancreatitis is highly variable and the surgical procedures employed are many. The principal reasons for electing surgery and the procedures employed may be briefly outlined as follows:

(1) *To correct associated biliary tract disease*—cholecystectomy with exploration and drainage of the common bile duct or the latter alone if the gallbladder was previously removed.

(2) *To remedy complications resulting from pancreatitis:*—

(a) for jaundice resulting from narrowing of the common duct—cholecystojejunostomy or choledochoduodenostomy;

(b) for chronic duodenal obstruction—gastroenterostomy;

(c) for recurrent gastrointestinal hemorrhage—partial gastrectomy;

(d) for pancreatic cyst—internal drainage by means of anastomosis with the stomach or intestine or by means of external drainage with marsupialization;

(e) for pancreatic abscess—external drainage.

(3) *To relieve persistent pain:*—

(a) external bile drainage (cholecystostomy or choledochostomy) or internal bile drainage (cholecystojejunostomy or choledochoduodenostomy);

(b) pancreatolithotomy—when the larger ducts are obstructed by calculi which can be readily reached.

(4) *To relieve pain and disability which persist in spite of the foregoing measures:*—

(a) unilateral or bilateral dorsal sympathectomy and splanchnicectomy;

(b) ligation of the pancreatic ducts;

(c) partial pancreatectomy—when the disease is limited chiefly to the head or tail;

(d) total pancreatectomy—when the entire pancreas is severely diseased.

INTESTINAL OBSTRUCTION IN HODGKIN'S DISEASE

Case Report

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Since Hodgkin first published his account "On Some Morbid Appearances of the Absorbent Glands and Spleen" in 1832, a vast literature has accumulated. To the bizarre, diverse and even at times the controversial symptomatology, we desire to add this case report.

The patient, Mrs. A. J. R., a white female of 35 was admitted to the Brownwood Memorial Hospital for surgical intervention for "Cystic Ovaries". Her previous medical history is noteworthy in that a report from a pathologist on a resected (?) ovarian cyst is stated to be endometriosis (Operation 5 years ago).

On admission, the presenting symptoms were pelvic and lower abdominal pain and discomfort and slight fever. She had been having a low grade fever for the past 2½ months and had been studied previously elsewhere but no definite diagnosis had been made. The physical examination indicated "Adnexal Pathology" and nothing else. There were no palpable glands. C.B.C., urinalysis, blood Wassermann, and the agglutination studies for typhoid-paratyphoid and B. melitensis group of organisms were noncontributory. The roentgen examination of the chest was normal.

The patient was operated on 12/19/48 and the operative findings are listed as follows:—Cystic ovaries and adhesions to tubes and colon, these cysts contain dark bloody fluid.

A bilateral salpingooöphorectomy, appendectomy and severance of multiple adhesions was done.

The pathologist's report of the resected specimen reads as follows:—

Small serous cysts,
Follicular cysts and corpus luteum cysts of the ovaries,
Subacute perisalpingitis.

A few days after operation, the patient developed signs suggestive of intestinal obstruction and she was then sent to x-ray. Flat film studies of the abdomen were made 12/27/48. These studies demonstrated a mechanical type of obstruction in the region of the proximal jejunum. Intubation was started and operation was performed the same evening (I assisted at this, the second operation).

At operation, a distended loop of jejunum was found and decompressed by needle, a mass of inflamed and enlarged glands in the root of the mesentery and the ligament of Trietz was noted, and a few adhesions in the ileocecal area were broken up. The presence of brown-purple patches, about 3 cm. in diameter on the antimesenteric border of the small intestines was noted and commented on.

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Their appearance was markedy similar to that shown in photographs and colored drawings on endometriosis. The liver and spleen were grossly normal and at this operation no evidences of extralumenal or intralumenal tumefactions to account for the obstruction could be found, other than the glands noted above. No glands were removed, as the patient's condition was desperate.

The patient's postoperative course was very stormy. She ran a spiking fever and the possibility of perinephritic or subphrenic abscess was considered. However these diagnoses could not be confirmed by adequate studies.

During this time, the patient received supportive medications, including the antibiotics and whole blood. Following the second abdominal intervention, eosinophilia and leucocytosis were noted on several occasions. These findings with the enlarged mesenteric lymph nodes led to a search for superficial nodes. A cervical lymph node was secured and biopsied. The pathologist's report (Terrell's Laboratory, Ft. Worth) follows:

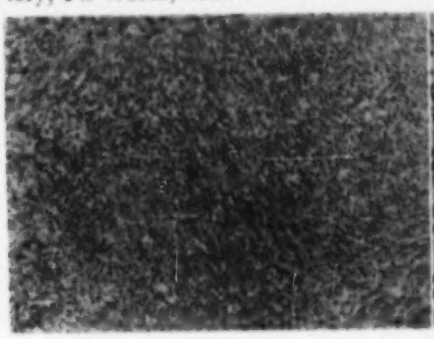

Fig. 1

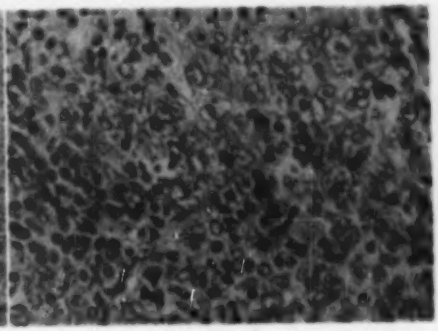

Fig. 2

Gross Findings:—The lymph node measures 2 x 1.2 x 1.2 cm. It has a gray smooth intact capsule. Section shows the normal lymphoid tissue to be apparently replaced by homogeneous firm gray tissue. Cortex and medulla cannot be distinguished grossly.

Microsections of the lymph node show the normal architecture to be completely obliterated by cellular tumor-like tissue. This tissue is composed of several different kinds of cells of which the predominating is a large, slightly elongated cell with hypertrophied and hyperchromatic nuclei. The nuclei tend to be vesicular in appearance having coarse chromatin material. Mitotic figures are seen frequently. Some of the cells have even larger darker staining nuclei which are sometimes multilobed, presenting the characteristics of Dorothy Reid cells. Scattered among these large reticulum type cells are seen many infiltrating lymphocytes, plasma cells and a few eosinophils. There is present a moderate amount of reticular stroma between the tumor cells and this is most marked in the central portions of the node where areas of fibrosis are seen. The capsule is thickened due to fibrous proliferation and contains a few infiltrating lymphocytes.

Pathological Findings:—Hodgkin's Disease of cervical lymph node.

The patient's course was progressively down hill. She continued to have fever, was jaundiced from time to time, became increasingly anemic despite repeated transfusions, had periods of abdominal distention and finally expired the first week of April, 1949.

Once the diagnosis of Hodgkin's Disease was made, she was given protracted and fractionated external radiation, first to the cervical glands, which appeared very rapidly after the biopsy, and then later to the inguinal glands when these showed up, and of course to the intraabdominal nodes. The external radiation succeeded in making the superficial cervical and inguinal nodes shrink, recede and disappear. They recurred, however. What the effect of the radiation was on the intraabdominal nodes could not be observed directly. However, significant relief of the abdominal distention was observed.

A postmortem examination could not be obtained.

Tumors of the lymphoblastoma group have been shown to cause obstruction of the small intestines when they are located intralumenally, and also extralumenally but intramurally[1,6,8].

Reports of obstructive jaundice caused by Hodgkin's Disease of the portal glands have appeared in the literature and there are many records of Hodgkin's Disease of the stomach, liver and spleen. In addition, there are a number of extrinsic lesions causing intestinal obstruction by pressure, e.g., fibroid uteri[2]. It is true that in this case, the presence of adhesions in the ileocecal area points to an immediate proximate cause. However, the enlarged and inflamed glands at the site of the obstruction in the jejunum must be regarded as the main causative factor in the findings at the roentgen examination and at operation.

An editorial in the Journal of the American Medical Association (Feb. 5, 1949) may lead us to infer that superficial lymph node involvement precedes deep lymph node involvement[3]. Muir[7] states that lymphadenoma usually starts first in the cervical glands and spreads to other groups, but in a fair percentage of cases the retroperitoneal glands are first involved and from these there is an extension to the mesenteric glands, inguinal glands, etc. Boyd[5], in his textbooks on pathology, is relatively noncommittal on this point.

Slaughter, Craver and Symmers[4] believe that the disease commonly begins in the internal lymph nodes, possibly having gained access to them through the gastrointestinal or respiratory system. Extrahepatic glands, causing pressure on the biliary system are thought to be responsible for the jaundice seen in 3-8 per cent of Hodgkin's Disease patients.

Summary:—The occurrence of intestinal obstruction in Hodgkin's Disease is noted and a case discussed. In this instance, superficial lymph node involvement was later than deep node involvement.

I am indebted to Dr. R. G. Hallum for permission to cite the clinical aspects of this interesting case.

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EMERGENCY GASTRECTOMY IN THE SURGICAL TREATMENT OF PERFORATED PEPTIC ULCER

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The principles and methods of treatment of acute perforated peptic ulcer have shown rapid strides in the last 10 years. This is easily understandable in view of the recent advances in modern surgery, anesthesia, chemotherapy and in the better knowledge of normal and pathological physiology. While a few physicians tend toward conservative treatment[1,2], the majority of surgeons agree that perforation is a catastrophe which warrants immediate surgery. Recently there has been considerable discussion among surgeons as to the type of surgery that should be performed. Most surgeons still adhere to the conventional method of simply suturing the ulcer and defer any further surgery for a later date. However, there are a few surgeons, here and abroad, who believe in an immediate more radical surgical procedure[3,4,5].

The better understanding of the morbid pathology of patients with perforated peptic ulcer and follow-up studies of these cases will give a proper answer to the problem.

The picture of the patient with acute perforated peptic ulcer is certainly dramatic; the initial clinical symptoms of perforation are usually abrupt. An apparently healthy patient with no complaints is suddenly seized with a sharp, midepigastric pain, with radiation to the shoulder and back, associated with painful, shallow breathing and boardlike rigidity of the abdomen. Although the pulse is normal and the blood pressure is rarely diminished, the patient is cyanotic, perspires profusely and is in agony. While some do not consider this condition as a stage of true shock[6], others feel that we are dealing with a type of neurological shock. One to three hours after the onset of this abdominal catastrophe pain often disappears and the patient feels quite well but, after six to eight hours, the pulse begins to rise, the abdomen shows lack of peristalsis, progressive distention and diffuse tenderness. The tongue is dry and coated. The picture of peritonitis with all its symptoms develops. What is the mechanism of the above mentioned signs and symptoms and how can this condition be corrected?

Formerly, it was believed[7] that the viscera themselves were insensitive to pain and that the visceral nerves contained no pain fibres. We know now that this belief is erroneous. The viscera have the same type of nerve fibres as the skin, but they are fewer in number. An adequate stimulus can initiate a true visceral pain via the sympathetic nerve fibres[8]. In addition, the sensation of pain is intensified if the visceral mucosa is inflamed, congested and hyperemic[9],—conditions quite common during perforation. Besides, there are at least two other mechanisms

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responsible for the origin of abdominal pain. First, the referred pain, in which impulses are carried via the sympathetic and cerebrospinal nerve fibres, and secondly the peritoneocutaneous reflex of Morley[10], which carries impulses via the cerebrospinal nerves only. The first mechanism is mainly responsible for the inhibition of peristalsis and the profuse perspiration with loss of water and salt; the second mechanism is responsible for the abdominal wall rigidity and the decreased activity of the diaphragm and the rest of the respiratory muscles. This results in poor oxygen exchange with cyanosis and secondary congestion of the lungs. The last factor predisposes pneumonic processes, a very common complication of perforation.

The first two mechanisms probably initiate the alarm reaction, and as a part of the general adaptation syndrome[11] stimulate the pituitary gland to release ACTH hormone. ACTH in turn stimulates the adrenal cortex, which releases its steroid hormones and a period of recovery takes place manifested by temporary clinical improvement.

Immediately following the perforation and the leakage of gastric contents, peritonitis develops. Since there are practically no bacteria in the stomach because of the bacteriocidic action of the hydrochloric acid, this type of peritonitis is purely chemical[12]. It inhibits peristalsis and gastric secretion, which results in decreased acidity, thus removing the barrier for bacterial growth. After six to eight hours the bacteria from the mouth and the intestinal tract spread to the stomach and peritoneal cavity. These statements have been proven by bacteriological studies[13]. Due to the fact that gastric contents are carried along the root of the mesentery, pain may be produced in the right lower quadrant and this may simulate the picture of a ruptured appendix. With the onset of bacterial peritonitis, the temporary period of improvement (so-called "countershock" in terms of alarm reaction) is gradually replaced by a stage of exhaustion with rapid downhill course.

Another very important problem for the surgeon to cope with, is the fact, that the ulcer patient usually suffers from malnutrition. This in turn results in secondary anemia, hypoproteinemia and low vitamin levels. This factor should not be overlooked, because the results of the postoperative recovery—as far as the wound healing and the resistance to infections are concerned—depend largely on the body reserve.

It is true that the body itself attempts to regulate the acid base equilibrium and the dehydration by blowing off carbon dioxide and drawing fluid from the interstitial spaces. Since this is not sufficient, the pathologic physiological changes and biochemical derangements described above are to be corrected before any type of treatment is decided.

First the pain sensation must be abolished by intravenous or intramuscular administration of morphine or its derivatives, then parenteral fluid therapy is started. Dehydrated and undernourished patients require: blood, plasma, sodium chloride and water. It is our tendency to correct the sodium and chloride ion depletion before operation, and then withhold sodium chloride administration for the first 24 hours following surgery. The postoperative water requirements may be

covered by 5 per cent glucose in distilled water. This step is in agreement with the phenomenon described by Coller[14] and his associates as "postoperative salt intolerance".

Whole blood is usually given just before and during operation in an attempt to restore the hematocrit value to normal levels. Plasma and amino acids are given after operation to restore the plasma protein level. It is difficult to determine how much fluid is to be given, but no harm can be done if clinical judgment is used. The daily salt requirement is given and rapid infusions of large amounts of fluids are avoided. Signs of paralytic ileus and abdominal distention are successfully treated by passing a Levine or Miller-Abbott tube with continuous or intermittent suction as suggested by Wangensteen.

In the treatment of the secondary peritoneal infection[15,16] following contamination from the alimentary tract, a new era was introduced with the use of the sulfonamides and antibiotics. When the sulfonamides and penicillin were used postoperatively in patients with perforated peptic ulcer, the postoperative febrile period was 6.3 days, whereas, it was 8 days when penicillin alone was used, and 12.4 days when only sulfadiazine was administered. Although the infection is usually mixed, the predominant causative organism is most often the colon bacillus. Penicillin in massive doses adequately controls the infection, without inhibiting the colon bacillus which can be combatted by the natural resistance of the peritoneum. If the results be unsatisfactory after the administration of penicillin for 24-36 hours, the peritonitis is probably due to the colon bacillus, and streptomycin and the sulfonamides should be started.

Since the prognosis is greatly influenced by the time elapsed between the perforation and the operation, the use of chemotherapy does not justify a delay in the surgical intervention.

The above pathological consideration should serve for a better understanding of the treatment. In view of this, it is apparent that conservatism is not justified for the following reasons:

1. The danger of generalized peritonitis is not avoided by the use of chemotherapy and decompression.
2. If there is leakage of solid gastric contents into the abdominal cavity, a subphrenic abscess may develop.
3. If healing does take place, reperforation is likely to occur shortly thereafter.
4. The main pathology (the ulcer) remains with its associated symptoms unchanged.

Since surgery seems to be the advisable treatment, two points should be considered: the immediate results and the late results. To make the picture clearer, two reports dealing with the follow-up studies of patients treated for perforated peptic ulcer by the simple method are presented. The first report deals with 101 cases[17] treated from 1928 to 1945. Of this group secondary operations were carried out in 17 cases for reperforation, hemorrhage, obstruction and intractable pain. It is quite clear that although the immediate results of surgery for perforated ulcer

TABLE I

	Year	Age & Sex	Occupation	Location of ulcer	Duration of perf.	Ulcer history	Operation	Duration of oper.	Drains	Anesthesia	Days in Hospital	Complications	Results
1	46.	M. 45	patrolman	duodenum	4 hrs.	none	Polya	1 hr. 5 min.	none	spinal	10	none	cured
2	46.	M. 21	clerk	stomach	3 hrs.	4 yrs.	Polya	1 hr. 20 min.	none	spinal	19	none	cured
3	47.	M. 20	manufacturer	duodenum	24 hrs.	none	Polya	55 min.	one	spinal	13	none	cured
4	47.	M. 44	laborer	stomach	4 hrs.	1 yr.	Polya	1 hr. 10 min.	none	spinal	8	none	cured
5	48.	M. 28	laborer	duodenum	6 hrs.	3 mos.	Polya	1 hr. 35 min.	one	spinal	13	none	cured
6	48.	M. 49	laborer	duodenum	4 hrs.	13 yrs.	Mayo	1 hr. 10 min.	none	spinal	11	none	cured
7	48.	F. 38	packer	duodenum	5 hrs.	3 mos.	Polya	1 hr.	none	spinal	10	none	cured
8	48.	F. 54	housewife	duodenum	5 hrs.	2 yrs., 4 mos.	Polya	1 hr. 10 min.	none	spinal	14	none	cured
9	49.	M. 41	bricklayer	stomach	2 hrs.	10 yrs.	Polya	1 hr.	none	spinal	13	none	cured
10	49.	M. 25	longshoreman	duodenum	1½ hrs.	none	Polya	50 min.	none	spinal	10	none	cured
11	49.	M. 62	salesman	duodenum	6 hrs.	2 yrs.	Polya	1 hr.	none	spinal	11	none	cured
12	49.	M. 48	laborer	duodenum	1 hr.	7 mos.	Polya	1 hr. 10 min.	none	spinal	15	none	cured
13	49.	M. 30	supervisor	duodenum	5 hrs.	6 mos.	Polya	1½ hrs.	none	spinal	11	none	cured
14	49.	M. 63	oiler	duodenum	3 hrs.	3 yrs.	Polya	1½ hrs.	none	spinal	15	none	cured
15	49.	M. 60	laborer	duodenum	24 hrs.	none	Polya	1 hr.	none	spinal	11	none	cured
16	49.	M. 23	clerk	duodenum	8 hrs.	2 yrs.	Polya	55 min.	none	spinal	7	none	cured

were good, the late results were interpreted as poor. In spite of that a conclusion was drawn, that the perforation should be closed by simple suture and only when the simple suture causes an obstruction, should it be combined with gastroenterostomy. In the second group[18], 100 cases of perforation were followed-up for the average period of 3 years and 4 months. The results were described as excellent in 28 cases, fair in 22 cases and poor in 23 cases. About 58 per cent of the cases continued to show ulcer activity by x-ray examination. Secondary surgical procedures were performed in 11 patients. In the same series, there were 12 additional patients who were absolutely advised to have a subtotal gastric resection, but most of them refused the operation. In other words, pooling the results, 45 per cent of the cases had an unsatisfactory outcome and 23 per cent required additional surgery.

Keeping in mind: 1. The poor late results of conservative surgery; 2. The technical difficulties in reexploration of the abdominal cavity; 3. The possibility of malignant degeneration of the peptic ulcer; 4. The difficulties in obtaining consent for a second operation; 5. The not common, but still occurring immediate complications of conservative surgery in the form of reperforation and bleeding, and realizing our present advances in: 1. Maintaining the fluid equilibrium; 2. Preventing the peripheral vascular collapse; 3. Controlling secondary infection and 4. Covering nutritional requirements, we feel that immediate gastric resection for perforated peptic ulcer is more justified.

Case Reports

From January 1st, 1946, to December 31st, 1949, sixteen cases of perforated gastroduodenal ulcers were treated with subtotal gastrectomy.

In our series, there were 14 males and 2 females, the average age was 40.6 years, the oldest was 63 years old, the youngest 21 years.

There were 13 cases with a history of ulcer or gastrointestinal complaints, suggesting the presence of an ulcer. Three patients (1, 3, 10) had absolutely no ulcer history and the first symptom of an ulcer was the perforation.

Two patients (2, 4) had a previous perforation, which was treated with the simple suture method.

There were 3 gastric and 13 duodenal ulcers. None of the ulcers was malignant.

The time between the perforation and the operation was 6 hrs. or less in 13 cases, one patient perforated 8 hrs. prior to surgery and two (3, 11) had a 24 hr. interval. The average time was 6.1 hrs.

The operation performed was, in all cases, a subtotal gastrectomy and in the cases of duodenal ulcer, the portion of duodenum which contained the perforated ulcer was also resected. The incision in all cases was an oblique xiphoumbilical. The gastrectomy was performed according to the technic of Polya in 15 cases and in one case the Mayo technic was employed. Usually ¾ of the stomach was removed. The gastrointestinal continuity was restored in 14 cases by a posterior gastroenterostomy. In only 2 cases was an anterior gastroenterostomy done. In 3 cases (10, 12, 13) an additional appendectomy was performed. The abdominal

cavity was washed in all cases with a sterile solution of sulfadiazine and some of the solution was left in. In only 2 cases was the abdominal cavity drained. The first case (3) had a history of perforation 24 hrs. prior to operation, the second case (5) had advanced peritoneal reaction with thick, cloudy fluid.

The average duration of operation was 1 hr. and 8.7 min., the longest time was 1 hr. and 35 min., the shortest 55 min.

All patients were operated on under spinal anesthesia.

The average hospital stay was 12 days, the longest being 19 days, the shortest, 7 days. The longest morbidity of 19 days in the hospital was noted in a case who was reexplored because of a previous perforation; the first one about two years prior to this operation was treated with simple suture technic. It is significant that the two cases with a 24 hour history of perforation prior to surgery (3, 15) had an uneventful recovery in 13 and 11 days respectively.

In this series none of the patients died. There were no complications.

Summary

1. Present trends in the treatment of perforated gastroduodenal ulcer are discussed.

2. For the better understanding of the problem, mechanisms of clinical signs and symptoms from the physiologic-pathological standpoint are mentioned.

3. Correction of the morbid pathology is evaluated from the angles of fluid equilibrium and modern chemotherapy.

4. The opinion is expressed that conservative treatment is hazardous and not justified from the standpoint of immediate and late results.

5. Immediate results of simple suture technic are good, but late results seem to be poor. To support this, statistical follow-up studies are presented and analyzed.

6. Arguments for radical surgery are listed, technic and results of emergency gastrectomy for perforation of gastroduodenal ulcers are discussed.

Conclusions

Our series is too small for definite conclusions. It proves, however, that with the proper correction of the pathological changes and with the use of modern chemotherapy, subtotal gastrectomy is a safe procedure and the most effective treatment for perforation of gastroduodenal ulcers. It gives, according to the present knowledge, the highest percentage of complete cures and has less than 1 per cent recurrences.

We would reserve the simple suture technic only for cardiac or diabetic patients and patients with grave and persistent peripheral collapse.

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THE PATHOLOGY OF JAUNDICE

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The opening chapter in a consideration of the jaundice problem might profitably be preceded by a short review of the erythrocyte's life cycle. It is from hemoglobin that bile pigments are formed and excessive accumulation of these pigments in the blood stream and body tissues constitutes the symptom known as jaundice and indicates an abnormality in the metabolism of bile.

It has been estimated that the life span of a healthy red blood corpuscle is from four to six weeks, after which it dies and is digested by specialized cells distributed widely throughout the body. If carmine is injected intravenously in the living animal, certain cells will retain the dye, notably the endothelial cells of the spleen, branched cells of the bone-marrow interstitial cells of the testes, reticulum cells of the lymph nodes and the Kupffer cells of the liver. Other endothelial cells, those lining the blood-vessels for example remain unstained. Tissues which retained the dye following vital staining were collectively designated by Aschoff as the reticulo-endothelial system, one function of which is to transform hemoglobin into bilirubin.

Hemoglobin is a complex chromoprotein composed of globin (a histone) and hematin which chemically belong to a group of substances containing four pyrrol

radicals and known as porphyrins. Hemoglobin then is an iron-containing porphyrin which is split by reticulo-endothelial activity to hematoporphyrin ($C_{34}H_{38}N_4O_6$) an isomer of the bile pigment, bilirubin. Conclusive evidence is available to prove that the liver is not essential for bilirubin formation.

After total extirpation of the liver, dogs rarely survive more than six hours, but in that period the tissues become intensely yellow and there is marked elevation of serum bilirubin. Similar results follow when splenectomy is performed on the hepatectomized animal indicating an extrahepatic and extrasplenic origin of bile pigment. Careful measurement of the bilirubin differential between the arterial and venous blood supply to any organ gives an accurate index of its bile production capacity. A splenic vein sample contains only moderately more bilirubin than does one from the splenic artery, whereas similar evaluations in relation to bone-marrow indicate this tissue to be the principal site of bilirubin formation.

The bile pigment is transported by the portal vein to the liver in the form of a bile-protein salt, bilirubin-proteinate having a high molecular weight. The molecule is too large for filtration through the renal glomerulus, an important diagnostic characteristic as the urine is always acholic with reference to this type of bile. Within the liver, the bilirubin passes from the interlobular veins through the hepatic cells into the bile channels and finally via the hepatic ducts to the gallbladder.

It has always been difficult to form a mental picture of the components of a hepatic unit, i.e., portal vein, Kupffer cell, hepatic vein, bile duct and liver cell.

McNee's well known diagram helps to simplify the matter (Fig. 1). The liver is regarded as consisting of a number of elongated tubules each with one closed end against the central (hepatic) vein and its lumen continuous with a bile canaliculus. Portal vein branches are situated between the tubules.

At the periphery of the lobule, the polygonal hepatic cells lining the bile canaliculi merge with the cubical cells lining the larger bile ducts. This is an ideal arrangement for securing intimate contact between the portal blood supply, the hepatic cell and the biliary system.

In its passage through the liver cell, the bilirubin-proteinate complex is broken down, liberating the smaller bilirubin molecule, which also becomes altered chemically and then passes into the bowel lumen. On reaching the large intestine, bili-

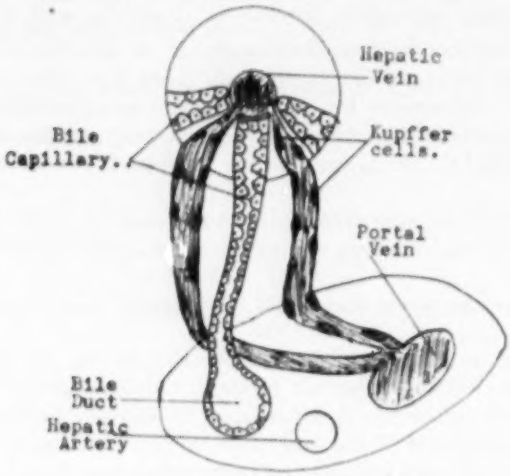

Fig. 1—Structure of liver lobule (McNee).

rubin is reduced by bacterial action to urobilinogen, a colorless compound, part of which is excreted in the feces as stercobilin. The remainder is reabsorbed from the bowel and returned to the liver (through the portal circulation) where the majority of urobilinogen is oxidized to bilirubin. A smaller portion continues unchanged through the hepatic vascular system from which it is excreted in the urine as colorless urobilinogen. On exposure to light, urobilin is formed and may readily be detected by Ehrlich's aldehyde test.

Various efforts at an omnibus classification of jaundice have been unsuccessful because they failed to include, on a basis of disturbed physiology, all types of icterus.

A logical and not original attempt in this direction is to consider jaundice as being either prehepatic, hepatic, or posthepatic. This takes into account the normal course of bile excretion and places the mischief either before, in, or after the liver.

Prehepatic Jaundice

When there is increased erythrocyte fragility, excessive quantities of bilirubin-proteinate are formed by reticulo-endothelial activity. This occurs in congenital and acquired hemolytic jaundice. This condition, particularly the congenital variety is associated with red blood corpuscles which hemolyze when exposed to an 0.75 per cent NaCl solution. Normally hemolysis does not start until a dilution of 0.45 per cent is reached. The increased quantities of bilirubin-proteinate are carried to the liver where the molecule is split and excessive amounts of bilirubin pass through the hepatic and bile ducts into the intestinal tract. Here bacterial activity produces large quantities of stercobilin and urobilinogen, excretion of the latter taking place in the urine.

Judicious application of laboratory tests will differentiate this type of jaundice. The icteric-index is elevated, stercobilin and urobilin reactions are strongly positive, but no bilirubin appears in the urine. It is an acholuric jaundice. A similar clinical picture is associated with, (1) hypersplenism in which the spleen destroys great numbers of erythrocytes and in (2) icterus neonatorum where a neonatal polycythemia in the newborn is reduced by increased hemolysis.

Recently Coombs has provided a valuable test for distinguishing congenital from acquired hemolytic jaundice. The surgical importance of this differentiation is associated with the poor results (85 per cent unimproved) accompanying splenectomy in the acquired type. Prognosis, following operation in the congenital variety, is so favorable that a method of correctly identifying and grouping patients clinically is most desirable. The highly significant feature of familial jaundice history is unfortunately too often absent in even a true congenital type and the patient may thus be denied the benefits of a life-saving procedure.

Coombs found that when a suspension of the patient's red blood cells is mixed with dilute antihuman rabbit serum, macroscopic agglutination of the cells may occur within one hour. This is designated as a Coombs' positive reaction and indicates acquired hemolytic jaundice. Failure of agglutination is a "negative reaction" and accompanies the congenital type.

Posthepatic Jaundice

This syndrome accompanies obstruction to the bile ducts *after* the bilirubin-proteinate has passed through the hepatic cell. The obstruction may be complete or incomplete. In the former, no bile enters the intestinal lumen, urobilinogen is not produced and no urobilin appears in the urine. The blood and urine bilirubin however are greatly elevated.

When duct obstruction is present but incomplete, small amounts of bilirubin enter the intestinal tract and urobilinogen tests remain normally positive although the quantity excreted in the urine is quite small. Proximal to the obstruction, the bile pigments accumulate in high concentration and under pressure in biliary channels. Regurgitation through the hepatic cells into the venous system occurs and there now appears in the blood increased amounts of bilirubin which was altered chemically by its original passage through the liver cell. When the serum concentra-

tion reaches 1:50,000, bilrubin appears in the urine where it gives a positive oxidative reaction with hydrochloric acid and hydrogen peroxide. Thus the presence of bilirubin in the urine differentiated simply and accurately, obstructive from hemolytic jaundice without recourse to the more involved Van den Bergh tests.

Hepatic Jaundice

During the early stages of diminished liver function there is often an accompanying jaundice, hemolytic in type. The hepatic cells become swollen, degenerate and many die so that they are unable to excrete even the normal amounts of bilirubin carried to them. Although the reserve potential of the liver is tremendous an infection or a toxin which seriously incapacitates large areas of hepatic tissue will be associated with retention at those points where normal passage of bile is disturbed. The liver as a unit may remain able to faithfully discharge its multiple functions uhtil recovery ensues, but the effects of focal liver dysfunction on bile metabolism will become manifest if the results of selected tests are judiciously interpreted. Assume that a sufficient amount of liver tissue has become defunct so that jaundice is produced. This may result from exposure to chloroform, toluene and its derivatives, arsenic, or the toxins of bacteria soluble in bile such as the pneumococcus. There is an increase of bilirubin-proteinate in the blood, the diseased hepatic cells are unable to excrete urobilinogen formed from the bilirubin in the large bowel and an increase in the urine urobilin occurs. The Van den Bergh reaction is of the indirect type.

The clinical confusion which can accompany the interpretation of laboratory tests in jaundiced patients with liver necrosis is not due to any fault of the tests but rather to a misappreciation of what is taking place within the liver itself. When, for example, the swollen liver cells cause a retention of blood bilirubin, the Van den Bergh test is of a pure indirect type. Should the cells at the same time occlude some bile ducts the element of regurgitant or obstructive jaundice is added and the Van den Bergh will show a biphasic reaction. After a few weeks, if the swelling diminishes sufficiently so as to allow the complete excretion of bilirubin but not enough to relieve the duct occlusion, the reaction will become a pure direct. The clinician, under these circumstances, is liable to declare the tests invalid whereas they reflect with great accuracy exactly what is taking place in the liver.

Rabinowitch has pointed out that a representative insight into the disturbed liver physiology associated with hepatic necrosis can be obtained by serial quantitative urobilinogen and Van den Bergh determinations. It appears that an increase in urobilinogen (urine) always precedes a rise in blood bilirubin, indicating that the sick liver cell is less permeable to the former than to the latter.

As the cells become more severely injured and less permeable both urobilinogen and bilirubin are retained, and there is a quantitative increase of both in the blood. Eventually, should obstruction become complete, no bilirubin is excreted into the intestine, urobilinogen is not formed and its blood concentration drops rapidly. Bilirubin values, however, continue to increase. When healing takes place urobilinogen is produced from the initial quantity of bilirubin which passes into the bowel so

that among the first signs of recovery in toxic hepatitis is the appearance of urobilinogen in the urine.

The number of available liver tests indicates the multiple functions of this organ. Probably the best that can be done clinically is to select two or three procedures which mirror, with reasonable accuracy, the inner disturbances of hepatic physiology. In addition to the blood and urine bile determinations already indicated, the following tests may provide pertinent information.

(1). Cephalin flocculation reaction.

(2). Thymol turbidity.

(3). Prothrombin time and response to Vitamin K.

(4). Galactose tolerance.

(1). *Cephalin Flocculation Reaction:*—This test was introduced by Hanger in 1939 and depends on the presence of an abnormal lipoprotein in the blood serum of patients with parenchymatous hepatic disease. The amount of flocculation is directly proportional to the degree of intrinsic liver damage. Flocculation degrees vary from 1 to 4, grades 3 and 4 indicating definite hepatocellular dysfunction.

(2). *Thymol Turbidity:*—In 1944 Maclagen proposed this test which has proven to be one of the most accurate in the laboratory differentiation of obstructive and intrahepatic jaundice. In the presence of the latter, turbidity readings greater than the maximum normal of 4 are noted. The test is performed by mixing serum with a saturated solution of thymol at pH 7.8, and comparing the turbidity with Kingsbury standards.

(3). *Prothrombin Time and Response to Vitamin K:*—Prothrombin is a protein produced by the liver in the presence of adequate amounts of Vitamin K. A prothrombin deficiency can therefore be caused by hepatic insufficiency or by lack of this vitamin. It appears that bile is required for the absorption of Vitamin K from the intestinal tract and also that the normal bacterial flora there is essential for its manufacture. In obstructive jaundice diminished amounts of bile are excreted into the duodenum, Vitamin K is not absorbed and the prothrombin time is prolonged. Therefore in a patient with jaundice and an increased prothrombin time, the administration of Vitamin K will be followed rapidly by a normal prothrombin test if the jaundice is obstructive in type. When primary hepatocellular damage is responsible for the prothrombin abnormality, Vitamin K therapy is ineffective. It is worth mentioning in this connection that attempts at intestinal sterilization by antibiotics especially streptomycin are often followed by prothrombin deficiency due to the removal of bacteria essential for Vitamin K production.

(4). *Galactose Tolerance:*—Galactose is a monosaccharide which is converted to glycogen by the liver and is not metabolized as such by any extrahepatic organ. The test dosage may be given orally or intravenously, the latter being preferable to eliminate possible faulty absorption from the intestinal tract. In the presence of hepatocellular degeneration, more than 20 mg. per cent of galactose is present in the

blood 75 minutes after the intravenous administration of 1 c.c. of a 40 per cent solution per kilogram of body weight.

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POSTOPERATIVE FETOR ORIS

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The presence of halitosis may be a distressing complaint of a patient following surgery. In severe cases, the patient will complain of having a foul breath. In less pronounced instances the nurse will call attention to the fetor oris.

It must be remembered that the etiological factor of halitosis may have been present prior to operation. However during the preoperative period it may pass undetected. During the period of convalescence following operation, when the patient is concentrating on his physical health, the previously undetected odor becomes noticeable.

The commonest preoperative conditions which may cause an accentuation of fetor oris during the postoperative period are:

1. Pyorrhea alveolaris (almost always noted in patients past middle life).
2. Dental caries.
3. Atrophic rhinitis.
4. Bronchiectasis (may become more pronounced after general anesthesia).
5. Vincent's angina.

In patients with the above mentioned pathology, oral dehydration, which occurs frequently after operation, aggravates the preexisting foul odor.

Fetor oris which was not present before operation and occurs suddenly after major surgery may indicate the onset of a complication.

Postoperative complications associated with moderate or severe halitosis are:

1. Intestinal obstruction (may be fecal in type in low bowel obstruction).
2. Peritonitis.
3. Acute gastric dilatation.
4. Fecal impaction.
5. Lung abscess.
6. Postoperative parotitis.
7. Subphrenic or hepatic abscess (especially when it pierces the diaphragm).

The primary treatment of fetor oris secondary to the causes mentioned is, of course, to eliminate the cause. Immediately following a major operation this is not always possible. Often conservative measures, as employing intubation for intestinal obstruction and antibiotics for peritonitis and abscesses, do not eradicate the etiological agent immediately. Moreover patients with disease of the oral cavity, as pyorrhea, are usually treated after convalescence. Hence active treatment of the etiological agent must be delayed in most instances. For this reason palliative measures must be instituted.

It has been an efficacious practice to have the postoperative patient masticate chewing gum following surgery. This stimulates the salivary glands to activity. The glandular secretions keep the mucous membranes of the oral cavity moistened which lessens the fetor oris.

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Another method is to employ oral antiseptics to freshen the mouth and lessen the halitosis. One of the most satisfactory oral antiseptics is stomaseptine. It has been employed by many postoperative patients with excellent results. In addition to its antiseptic qualities, it is refreshing to the patient. For this reason many patients desire to use it as frequently as every hour.

Summary

1. Attention is called to halitosis as a distressing symptom after major surgery.

2. Etiological factors may exist before operation or may develop after surgery.

3. The sudden onset of fetor oris following an operation may indicate the beginning of a complication.

4. Eradication of the halitosis depends upon the correction of the etiological agent.

5. When this is not possible temporary measures aimed at the improvement of oral hygiene often lessens the fetor oris.

CORTICODIENCEPHALIC GASTROINTESTINAL SYNDROMES IN EPILEPTICS*

(Part XII)

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Case Reports (Continued)

C. P., an adult male patient was admitted to the institution in January, 1938, complaining of grand mal fits that are preceded by an aura of pain in the stomach situated at the left side of his navel which radiates up towards his head following which he feels a cold chill coming on him. He sometimes becomes violent and destructive before and after spells. Due to his attacks of abdominal pain, an appendectomy was performed upon him in the course of one of his spells when the abdominal algoleptic paroxysm was not associated with the usual convulsive fits.

His family history was negative for nervous and mental diseases. He was born in October, 1916. The delivery was normal and he weighed ten and a half pounds at the time of birth. He was breast fed, and teething began at nine months of age, while walking commenced at one year of age and talking, at one and a half years. He entered school at the age of six years and his scholastic progress was fair. He sustained a severe injury to his head at the age of two and a half years, when he was struck with a hoe. The records state that his injury occurred at 2½ years while the patient claims that at four years of age he sustained the above injury when he was taken to the hospital in an unconscious state. The appendectomy was performed upon him either during the period of his petit mal attacks which he suffered from for a long time before his actual convulsive type of seizures started, or later. The first epileptic attack occurred when he was 12 years old and the second spell took place one month later. On admission no other positive findings were made except that he was a moron having a mental age of 9.33 and an intelligence quotient of 58. The patient was examined by us a short time after admission when upon superficial inspection he seemed to be well developed as stated.

Upon detailed study the following findings were made. Although his gait was normal, there was a slight positive Romberg on his left side. Diadochokinesis though normal was carried out rather slowly. There was some diminution of muscle strength in some parts of his body and his general motor activities were

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definitely slowed. There were no other abnormal involuntary movements except tremors of his extended fingers. The following were the reflex findings. All the tendon reflexes in the upper extremity were exaggerated to three minus with the exception of the right triceps which was two minus. The left patellar and achilles were weaker than those of the right side. The plantar skin reflexes did not react at all. The right abdominal skin reflex was greater than the left one, and both cremasteric reflexes were absent. The combination of unilateral ataxia of the static type with reflex changes in the same individual again needs physiological comment. Throughout this manuscript the name of Muskens was mentioned in various connections dealing with the above two problems. Although Muskens did elaborate anatomical work upon the vestibular system, dividing the main nucleus into three parts and connecting them with the ocular and other cranial nerves, with the medial longitudinal fasciculus and with the inferior olivary body thus creating a system of tegmental and reticular fasciculonuclear structures establishing a basis for eye and head movements in the form of alternating reflexes, his work forms only a small fraction of the physiology of this system, when compared with the brilliant advances made along the same lines by R. Lorente de No. It is on the basis of the work of the last mentioned, that one can see the relationship between coordinative phenomena and reflex changes and finally tonus and rigidity as they apply not only to skeletal muscles but to those of the gastrointestinal system as well. It is on the basis of this as explained in experiments with nystagmus that Lorente de No elucidated the rationale for the existence of permanently pulsating systems. If the diencephalon contains them, then it is easy to understand the mechanism behind hypothalamic decerebrate rigidity as proposed by Thiele, Weed, Sherrington, etc., as related to organic signs of neurosomatic deterioration which starts with ataxia, etc. and ends with rigidity. Although there were no frank positive pathological reflexes, the above reflex changes are definitely positive indicating injury to both sides of the brain. The patient's pupils reacted sluggishly to light with a temporal pallor on fundus oculi examination but the visual fields were essentially normal.

The above case is an example of many similar patients whose epilepsy is the result of injury to the brain and in whom there are definitely positive neurological findings associated with mental defect. In this particular case, there is no question that the brain injury, though not extensive enough to produce a paralysis, was of sufficient gravity to affect not only parts of the cerebrum responsible for the production of convulsion but also such parts, within the brain, that produced not only an aura of abdominal pain but also the paroxysmal attacks of violence and destructiveness. Whether the manic periodic symptoms be localized in the frontal lobe in view of the absence of symptoms pointing toward the involvement of other lobes of the brain, or not, we cannot commit ourselves at present.

In the above patient we stressed the aura. We had a special reason for that. We stated that the abdominal pain was located near the navel. If we had taken pains to question every patient with regard to the actual location of the pain and as to whether radiation was present or not, our data would be much more exten-

sive. We realize fully well, that radiation into the groin or to the back, might disclose the actual cerebral representation of the visceral contractions. The abdominal pain might furthermore not be gastrointestinal in origin if the exact nature of the abdominal process responsible for the pain is determined by laparotomy. For the present we will however satisfy ourselves with merely presenting the data as offered to us directly and from the records since the proof which we are anxious to establish is that the lesion is cerebral. If the pain points toward a special viscus, we will utilize terms such as gastralgic, duodenalgic, ilealgic, colalgic, hepatalgic aura, indicating that the pain refers approximately only, to the above situations. Another patient who presented an aura of abdominal pain uncomplicated by other symptoms, was the following.

A. K. was an adult female who was admitted to the institution complaining of fits in which she falls to the right and which are preceded by the gastrointestinal aura.

Neurological examination showed a patient with a peculiar attitude, which is evident mostly on walking, during which there are no definite automatic associated movements. Otherwise this patient was normal, showing no motor, coordinative, or muscle strength disturbances. The patient could not cooperate well enough for a detailed sensory examination. Her tendon reflexes were essentially normal, except that the patellars were exaggerated to the third magnitude. There was a definite facial asymmetry with a questionable lagging of the left side of the face, while the tongue deviated slightly to the right. The examination of the fundi showed a blurring of the disc margin in the left eye, in which the choroid had a distinctly different appearance than that of the other eye. Stimulation of the carotid region produced no convulsive reaction. Our interest in carotid sinus stimulation is of course primarily the reproduction of the seizure pattern and its attendant train of gastrointestinal reactions. The ability to elicit such a reflex chain of reactions, depends upon a number of factors and according to the myoclonic shock theory of Muskens, there seems to be a temporal relationship between the results obtained at any particular time and the chronology of the last fit or series of fits in the individual upon whom the procedure is executed. Although we are primarily interested in the gastrointestinal accompaniments of the fit, the production of a seizure pattern consisting of head, eye and neck movements on a reflex basis, seems to be of great physiological importance not only in the light of the data presented in the Davison and Goodhart paper and in those of Foerster and Penfield, but particularly with reference to the work of R. Lorente de No on the vestibuloocular reflex arc. This reaction impresses one with the relationship between movement and reflex mechanisms extending from the cerebral cortex to the inferior olivary body. Our interest in head reflexes stems from the above.

No elaboration is necessary in this patient who is mentally deteriorated and suffers from convulsive seizures associated with an aura of abdominal pain, to show that the lesion responsible for all of the above symptoms, is located in the cerebrum. On previous occasions we have shown that in order for such a lesion to be located elsewhere in the brain other positive neurological findings such as

crossed paralyses, monoplegias, etc., are necessary. The above are definitely lacking in this patient.

J. W. is a male patient 61 years of age who presents a history of birth injury resulting in grand mal convulsions but accompanied by very few neurological findings. There is a definite aura of abdominal pain. The only positive findings are a marked edema of the nerve heads. In the above patient we have shown that in some cases very little of positive findings are necessary, so long as there is a definite history pointing towards organic brain disease. This, combined with the history of abdominal pain occurring in relation to seizures, is sufficient for one to localize the lesion responsible for this syndrome combination, in the cerebrum. At a later date, we expect to present a complete description of the method of differential diagnosis which we follow, to prove that some of the above patients are suffering from nonneoplastic brain lesions. We shall do so particularly in patients whose findings are especially suspicious of the presence of increased intracranial pressure. This is encountered in cases in whom the findings upon eyeground examination indicate differentiation between brain tumor, uremia, diabetes mellitus, systemic and renal disease, multiple sclerosis, spinal disease, syphilis of the brain, etc., etc. The following two cases, although not showing extensive neurological clinical signs nevertheless suffered from abdominal aura preceding epileptic attacks of many years duration.

J. M. was an adult male who presented the above syndrome combination. He did not suffer, however, from convulsive spells, but from petit mal attacks preceded by a gastralgic aura. The neurological findings, though few, were very important. Each eye showed a bilateral mild primary optic atrophy. The reader can therefore see from this case, how important it is to do a complete neurological examination, even though the findings seem meagre at the first glance. Every avenue of the routine neurological procedure should be checked, and every laboratory test including x-ray study, should be utilized, since an optic atrophy might be due to sinus or even systemic disease.

E. C. is an adult female 22 years of age who suffers from convulsions preceded by abdominal pain associated with a "feeling" in the head. The only positive neurological finding in her case, is an atrophy of the anterior ⅔ of her tongue. In the absence of anything else, the localization of the epileptogenic and gastralgic neurogenic focus is in the cerebrum.

In contrast to the above cases of pain syndromes in which localization was within the cerebrum, but not definitely in any restricted location, the following patient differs, in that the examiner can place the lesion responsible for the entire combination of symptoms, namely the convulsive as well as the gastrointestinal symptoms, in a specific tract. Furthermore, trophic changes are present and are associated with paralytic findings in the same parts of the body. The seat of the trouble productive of the above seems to be deep down in the cerebrum, near the internal capsule. Those fibres responsible for the trophic phenomena are probably such as might be directed from the internal capsule towards the mammillothalamic system or the preopticohypophyseal tracts around the third ventricle. In our

enthusiasm about the mammillothalamic pathways in relation to those of the dorsal longitudinal tegmental systems connected with the periventricular and preopticohypophyseal systems, we failed to impress the reader with the fact that the latter connects with telencephalic structures anteriorly. Although we are averse to conjuring up neuroanatomic ghosts, here and there it occurs to us that we are not professional anatomists and that a reviewer who has had more experience with degeneration experiments, might call our hand. Here and there we reminded ourselves that we were rather too enthusiastic about some authors. In the case of Bechterev for instance, it was claimed that he discovered one of the parts of the extrapyramidal basal ganglia. We read that in a reliable translated text from the Russian literature, but when we studied the Latin of Tiekemann we found the above claim rather shaky. While we are on the subject we wish it understood that we make few claims along therapeutic or electrophysiologic lines in epilepsy. We are primarily clinicians or plain doctors, if you will, who have made observations upon clinical neurological phenomena and insist upon reporting the same as we saw them. We saw a lot more, but the present trends call for psychodiagnosis rather than for reporting new reflexes or data on muscular atrophy. If Wartenberg disagrees with us, we will know it soon enough from his publications.

C. O. is an adult female who was admitted to the institution complaining of convulsions which are preceded by pain in the left side of the abdomen which radiates up towards the head. She was about 30 years of age, and her mental reactions indicated that she was not reliable for a detailed sensory evaluation.

The neurological examination disclosed a patient presenting a right hemiparetic gait accompanied by limitation of movements in the right upper and lower extremities. Muscle strength was somewhat diminished on the right side. There was a definite wasting or hypogenesis of the right hand. In addition to the above, there was a definite weakness of the right facial musculature and a moderate right choreoathetosis. Examination of the eye grounds revealed some involvement of the right optic nerve. The right areola was smaller than the left one, and the right side of the chest was narrower than the left. The above resulted in a right lumbar scoliosis and a dorsal kyphosis. The heart sounds varied in strength and the patient's blood pressure was 110 over 60. In regard to hemiatrophy of the chest in epilepsy or to hemihypogenesis of its contents in epileptics, we believe that if we have overstressed this condition we have done so only because we felt that the general health of the epileptic patient was affected because of it. In some of these patients with partial or total hemiatrophy, we noticed that the individual could not stand operative procedures as well as other epileptics. It was useless for us to try and convince the general surgeon or internist about this phenomenon, since text books do not recognize this entity. It seems that Francis Marion Pottenger is the only chest expert who gives credence to this phenomenon. For the above reason we stressed the work of Archambault and Fromm. In elaborating upon the work of the above we have no intention to throw our hats into the neurological ring and oppose such men as Professor Wartenberg and others. We feel that this eminent reflexologist could do justice to the subject of muscular,

osseous, or chondral and visceral atrophy. He has in fact been following along such lines in the past few years. It would be a greater feather in his cap if he established his stand on this subject in as firm a fashion as that skillfully executed by the great clinician Archambault.

The specific localization of the abdominal pain in the above case on the left side of the abdomen calls for a specific pain syndrome of a special somatotopic nature. The radiation of the pain towards the head adds another combination to the pain syndrome. We must therefore designate this as an ileojunal, or colalgic type of aura, combined with a cephalad radiation. It is interesting to note that this type of aura having such an extensive radiation, runs parallel with the type of lesion which is likewise extensive in a corporocephalad representative way in the cerebrum of this case. In spite of the above, we are at a loss to state as to where the localization of the pain actually might be. It is definite, however, that the choreoathetosis points toward the caudate nucleus while the limitation of movement and of muscle strength, indicate an involvement of the left internal capsule. From the above there is therefore no doubt that the seat producing the abdominal pain is a lesion in the left diencephalon or in the diencephalocortical pathways, or at least in the diencephalotelencephalic structures. It is also definite that the representations in the diencephalon of the viscera have an extensive somatotopic character. Both the visceralgia and the cephalalgia are therefore cerebrogenic. It will however have to be decided, whether the pain is nucleogenic, involving the nuclei of the dorsal thalamus or the lateral or ventral portion of the same. It might also be nucleogenic involving the caudate or preoptic nuclei. The pain, on the other hand, might be tractogenic or commissurogenic from neurones of the ventral thalamus which cross from the hypothalamic centers, passing through the mammillary commissure. In most cases of generalized abdominal pain aurae, we firmly believe this to be true, and most of our patients presenting abdominal aurae, suffer from general rather than local abdominal complaints.

In the presentation of the above cases we have invariably shown that since there is no possibility of brain stem involvement and that since the spinal cord and peripheral cranial nerves were excluded, the only structure remaining as a responsible factor in the neurogenic control of gastrointestinal function, is the cerebrum. Particularly in the last mentioned case, we took pains to show that the paralysis of the facial musculature was present on the side of the paralysis of the upper and lower extremities. This combination, would definitely place the lesion responsible for the visceralgic and convulsive, as well as the paralytic and trophic syndrome, within the cerebrum. In the following we intend to further fortify the above statements and offer some proof to show, that the lesion within the cerebrum occasionally produces descending degenerative changes, so that structures below it become secondarily affected. We see not only the clinical, but even the pneumoencephalographic evidence of this involvement of the cerebrocerebellar pathways. We cannot go into detail about the last-mentioned subject at present while dealing with the pain syndrome. We shall however attempt to deal with it later on.

D. M. M. is an adult female of 17 years who was admitted to the institution complaining of an aura consisting of pain in the abdomen which is succeeded by a grand mal convulsion.

The neurological examination showed an impairment of muscle strength on the left side constituting a left hemiparesis. In addition to this weakness, there was a slight limitation of movement. The gait disclosed a limitation of automatic associated movements in the left upper extremity. Coordination tests revealed a positive modified Romberg of the left side. There was in addition, a definite dysmetria on the left side, since the left finger to nose test, was abnormal. There was also an adiadochokinesis on that side. The above definitely indicates involvement of the cerebellar or cerebrocerebellar fibre systems.

In addition to that, there was a definite trophic syndrome suggesting a crossed involvement of the glandular neurocrine system, similar to that seen in some of the cases in the literature offered by Archambault and Fromm. The right breast was longer than the left one lacking glandular tissue, and the areola of the right breast was ovoid, and so was the right nipple thus showing, that, although the left side of the body was otherwise involved, the breast gland of the right side was affected. There was some wasting of the left upper extremity and of the left foot, thigh and the left chest. Coupled with the above, there was an atrophy of the left side of the tongue. In this case, we therefore observe a similar picture to that of the preceding one, in which the cranial nerves are involved on the side of affection of the upper extremity and the lower one. This indicates a cerebral lesion and excludes involvement of other parts of the brain. At present we cannot offer an explanation of the crossed neurocrine involvement, but shall do so in similar cases later on.

With the exception of the biceps, radial and achilles, the reflexes on the left sided extremities were greater than those of the right upper and lower extremities. Together with these, there was a positive Babinski on the left side. The patient did not seem to be reliable enough for a critical sensory examination. The atrophic syndrome in the cranial nerves other than the atrophy of the left side of the tongue, showed involvements of the optic division. The right nerve head was larger and paler than the left one, and the fundus oculi on that side showed tortuous and abnormally directed vessels.

The above findings therefore pointed to an involvement of the right side of the brain. That this is the case is borne out by the following analysis of the pneumoencephalograms, as shown in the views of Figure 71. All the views could not be included in this figure, but from the analysis, the reader can gather the essential features of the brain involvement. Anteroposterior view shows that the roof of the body of the right lateral ventricle is pulled outward. The ventricles are situated centrally. The right cerebral hemisphere is narrow at the top and there is considerable space on both sides of the falx with definite wasting of the hemisphere. In the course of preparation of this manuscript we have had inclinations to remark upon the value of pneumoencephalograms in detecting evidences of abnormalities of intracranial circulation, even in nonepileptics. This tendency grows out of our experience, particularly with fliers (Weingrow) in the recent war. We believe

that individuals preparing for a flight career, should be thoroughly studied pneumoencephalographically, wherever there is a history of severe head injury, no matter how far back that goes. During the war, electroencephalography hadn't reached the stage where small lesions that might produce meningeal obstructions, could be detected by the latter method. This applies, of course, more so, to cases who have engaged in many combat missions and have shown the effects of air, particularly nitrogen encephalopathy. The posteroanterior view of Figure 71 shows dilatation of the third ventricle. The convolution on the top of the hemispheres near the falx, shows a greater atrophy on the right side. The right hemisphere is narrower. The suboccipital view, seen on the extreme left of the figure, discloses such a layer of air over the tentorium, that is almost an inch in thickness, and extends across above both cerebellar lobes. This probably accounts for the positive cerebellar findings on neurological examination. The right lateral view

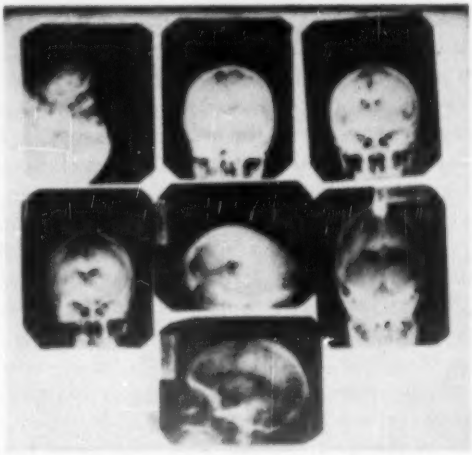

Fig. 71

shows a moderately dilated fourth ventricle and a great deal of air in the frontal, occipital and cerebellar regions.

In the above case analysis followed by the pneumoencephalogram, we observe that a paralysis of one side of the body may be followed by changes in the cerebrum of the opposite side of the brain. In other cases of this pain syndrome group, we have seen individuals with much more extensive paralysis and atrophy than was shown in this case. In our rather extensive experience with pneumograms of paralyses and atrophy of extremities in epileptics, particularly of the total hemiatrophy type, we saw moderate to marked, and in most instances the latter variety, of external hydrocephalus. Although we are engaged in the preparation of papers on the surgical and drug therapy resulting from a quarter of a century of experience, especially with the former, mainly by the senior surgeon (Fitch) of this neurosurgical team, our interests in hydrocephalus alone, has been

a most absorbing one. At present we have a manuscript in preparation dealing exclusively with the subject of internal and external hydrocephalus in epilepsy (Weingrow, S. M., Fitch, T. S. P. and Pigott, A. W.). There is, in our mind, no subject in the pathology of epilepsy that has been more neglected than the one dealing with hydrocephalus. Even in his monumental work on hydrocephalus Marburg; who was more conversant with the literature of neuropathology than anyone else of the present era, listed only about thirty or so, cases of epilepsy, in which the pathological aspects of hydrocephalus were reported upon. In our presentation of other syndromes we shall dwell on this subject and present proof of the above statements. At present we are merely interested in showing that the gastrointestinal pain syndrome group of cases, consists in involvement of the cerebral neurogenic pathways that control motor and secretory functions of the gastrointestinal tract. Whether such tracts are affected by lesions in the frontal lobe by secondary ventricular pressure of the lateral ventricles, or of other cerebral parts, remains to be seen. In the meantime, we wish to emphasize that the extent

Fig. 72

of the clinical findings does not necessarily parallel that of the pneumoencephalographic ones. This is seen in the autopsy findings of the case in Figure 72 and in the pneumoencephalographic findings of Figure 73 in a patient whose case we previously presented in the literature (Pigott, A. W., Weingrow, S. M. and Fitch, T. S. P., 1939 to 1940). In this case there were few positive clinical signs.

While occupied with the presentation of cases in the pain syndrome group above, it seems that we neglected to show how our presentation of the literature on the neurogenic control of gastrointestinal function applied to the material under consideration. We fully realize that we failed to mention how the work of Lennox and Cobb was related to that which we were offering. We stated, however, that abdominal pain in the form of an aura might suggest not only a sensory disturbance, but also a motor disturbance of the smooth musculature of the gastrointestinal tract. In this way it was not necessary to go back and review the work of the various authors which Lennox and Cobb cited. We took it for granted, that, in dealing with the cases of epilepsy that every phase of gastrointestinal involvement in epileptics, as mentioned by the above authors, apply to

the cases which we were discussing. What has been said above in regard to Lennox and Cobb, applies especially to our analysis of the literature as given by Donal Sheehan. In the cases of hemiplegia which we featured above, it was shown that almost invariably, the basilar portions of the cerebrum were primarily or secondarily involved, and because of this, it is not unreasonable to assume that the mammillotuberal and preoptic regions might contain lesions which were responsible for the gastrointestinal aura. Even if these localities were not directly affected, it is not unreasonable to assume that the hypothalamic areas or even the portions of the thalamus controlling visceral activities damaged by the cortical and ventricular pathology, might have been responsible for the gastrointestinal aurae preceding the convulsive episodes. That we are not incorrect in the above assumption, is moreover proven by the fact, that a good percentage of the cases which we offered, suffered from trophic or hypogenetic disturbances in the extremities. Such involvement is usually related to lesions in the diencephalon and particularly in the periventricular regions. That this is true has been shown by us in a thorough analysis of the literature as presented by Archambault and Fromm. We have therefore more than fulfilled our repeated promises, that our elaboration of the experimental literature by Beattie, Keller, Cushing, Karplus and Kreidl among others, would be justified by ample clinical data of our own (see Figs. 1-5).

In our efforts to prove to the reader that the gastrointestinal syndrome of pain associated with convulsions, was mediated in most instances by lesions within the cerebrum, we presented extensive neurological findings in a number of individuals. In other cases where the clinical material was not abundant, we concluded by the method of elimination, that it was the cerebrum and no other portion of the brain, that is responsible for the pain algoleptic syndrome.

We saw however from the presentation of the last few cases and particularly from their pneumoencephalograms, that in addition to the initial lesion responsible for the paralysis of the side of the body contralateral to the same, that other additional changes take place within the cerebral hemisphere. We showed for instance, how, a dilatation of the third ventricle as well as of the lateral ventricles, produces pressure upon various structures. Such structures upon which pressure is in this way exerted, are those which we had invariably emphasized in our presentation of the literature. The extensive array of authors whose experimental and clinical work on the mammillary region we emphasized is therefore automatically brought into our picture of clinical case presentation. What is being said of the authorities on the neurogenic diencephalic gastrointestinal control data applies also to our having dwelt on the literature covering these neuroanatomic structures with experimental and clinical evidences pointing to upsets in water and sugar metabolism. A dilated lateral ventricle, will invariably press upon the anterior nucleus of the thalamus and upon the caudate nucleus, and in a percentage of instances, upon the cingulate gyrus among other structures that we mentioned in the basket scheme of diencephalic autonomic neuroanatomy. In the presentation of the above cases, we did not have space to deal with an analysis of relationships between somatic paralysis, and the gastrointestinal aurae, as well as

other aurae such as the visual and auditory. We could not afford to deal as minutely in a critical way and to analyze interrelationship between the clinical symptoms and the neurological findings as did Archambault and Fromm. We left the above to the especially neurologically inclined reader, and organized our illustrations in such a way that approximate localization of lesions would be possible by anyone who might wish to do so. It involved a number of years of planning the organization of our illustrative material to meet with the above demand. For this reason, we have, in most of our illustrations, vertical lines representing coronal planes situated at various levels of the internal capsule. The reader can therefore pick any one of the hemiplegic, monoplegic, hemianesthetic or monoanesthetic cases, and, by combining the aura with the same, approximately place the lesion either towards the frontal, temporal or occipital portions of the cerebrum. If a visual

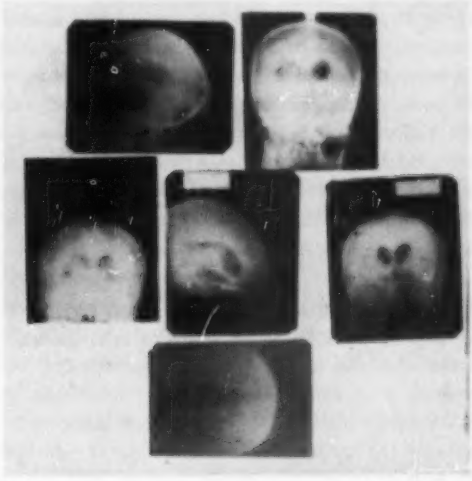

Fig. 73

hallucination is associated with a hemiplegia and gastrointestinal symptom, it is evident that the lesion would be located in the posterior section of the internal capsule and it may involve either one of the geniculates or part of the occipital lobe. Before terminating our discussion of the pain syndrome, we will present the following case as an illustration of a relatively complete neurological as well as pneumoencephalographic work-up. Extensive changes in many structures of a hemisphere secondary to pressure by a dilated ventricular system, should result in descending degeneration in tracts such as the corticothalamomammillary and others. Degeneration of the corticostriate, corticolenticular, and corticohypothalamic pathways should result in gastrointestinal disturbances according to Watts and Fulton. Such degeneration, if it involves particularly the lenticular nucleus might not unreasonably be accompanied by degeneration of the liver and thus account for the syndrome of hepatolenticular degeneration known as Wilson's

Disease. If such clinical entities not unanticipated by Watts and Fulton exist, and we have shown them not to be impossible, then we would be forced to call such syndromes corticopallidal hepatic degeneration, corticoputamenal hepatic degeneration or even corticocaudate hepatic degeneration. For the last mentioned combination we offer at least ten cases of athetosis indicating involvement of the caudate thalamic fibres or caudate lenticular complex. The material is here; the classification is up to the neurologist (see Figs. 30, 45, 46, 48, 52, 53, 54, 56-60).

E. J., an adult female, 18 years of age, was admitted to the institution in 1934, complaining of grand mal and petit mal seizures which are invariably preceded by attacks of pain in the abdomen. The spells were claimed to be of 17 years duration, the onset coming upon the patient when she was 16 months of age. Although the extramural diagnosis of this case was idiopathic epilepsy, it was evident that the history of paralysis shortly after or during birth, suggested that the diagnosis might be an incorrect one. Analysis of the aura in this case shows that this is not infrequently found in most patients. Although the average epileptic merely complains of a single symptom referring to the abdomen, it is usually not so. In her case, the pain in the "stomach" was associated with a funny feeling below the navel. She claimed that she experienced her pain in association with a feeling of tightness so that the abdominal muscles felt contracted. She claimed that the rigidity in the abdomen was followed by a similar condition in her extremities subsequent to which she became unconscious.

Her past personal history shows that she was paralyzed after birth for 16 months on the left side of the body. When the paralysis disappeared she began to suffer from epileptic spells. Other than the above her history is irrelevant except for her mother who died of a stroke at 55 years of age. Casual observation of the patient does not seem to show any extensive involvement, but a detailed neurological examination as presented herewith will show that she suffers from a residual of an old paralysis which has left definite evidence of failure in development of various parts of the muscles in the left side of her body.

Neurological examination showed that the patient stood better on the right than on the left side and that the left finger to the nose test was not properly executed. There was a definite difference between the right and the left hand the latter being somewhat wasted and the skin showed atrophic sclerodermal-like changes. The left foot was narrower than the right one. The tendon reflexes were equal bilaterally, all being two plus in magnitude except the left patellar which was greater than the right and the left achilles which was also greater than the right one. The left abdominal reflex was less than the right one although there was an appendectomy scar on the right side. Sensory examination was essentially negative throughout. Examination of the cranial nerves disclosed that the tongue deviated to the left and that there was a weakness of the left side of the face. There was some tilting of the head toward the right. Muscle strength testing showed a definite diminution on the left side. From the above it is evident that we are dealing with an individual who suffers from the residuals of an old birth injury to the brain. The involvement of the left side of the body is indicative of a

lesion or lesions in the right cerebral hemisphere. This patient was repeatedly checked and rechecked for the above neurological findings which were noted to be essentially the same. Eventually a pneumoencephalogram was done on her; and although she was classified as suffering from a moderate amount of general cerebral cortical atrophy, it was also shown, that there was definite evidence of incomplete filling of the cortical spaces on the right cerebral hemisphere. Thus it was felt that the lesion of a traumatic nature produced an arachnoiditis on the right side. The ventricular system was involved but not markedly. Such involvement was evidently secondary to the meningeal lesion of the right cerebral cortex.

From the above case we can see that in patients in whom the paralytic and atrophic findings are not extensive or readily noted, that a pneumoencephalographic procedure is of extreme usefulness in determining as to whether the lesion of the cerebrum involves the deeper portion of the brain such as the internal capsule or the cerebral cortex over an extensive area. If the neurologic clinician or neurosurgeon thinks that dealing with nonneoplastic lesions pneumoencephalographically is an easy matter we invite attention to the interpretation of the data in Figs. 71 and 72 comparatively and on a clinical interpretative basis. We have been only partially successful in our work of over a decade. Our team consists of two neurosurgeons and one roentgenologist. To accomplish the tracing of lesions on a corticocerebellar basis we had to do all the clinical neurological examinations ourselves. This consumed a great deal of our time away from our offices and at long distances from them. We were rather fortunate with institutional cooperation at that time since the roentgenographer of our team (Pigott) was the superintendent at the New Jersey State Village for Epileptics. Even in ordinary pneumoencephalography we resorted to the injection of tremendous amounts of air compared with the fluid removed in order to appraise the shock-like effects in cases of damaged corticodiencephalic vegetative centers. We never lost a case but came near it on such occasions. For these reasons we urge our readers to take our clinical observations on chest hemiatrophies seriously. We feel that a hemiatrophied lung goes a long way in undermining the resistance in epilepsy.

In addition to the usual pneumoencephalographic test we have executed in a limited number of patients, an additional procedure which is known as the subdural operative procedure. This was carried out in the above patient in November of 1938. A burr-hole opening was made on the right side of the skull by one of us, while an assistant removed some cerebrospinal fluid from the lumbar region. Incision of the dura permitted the entering of air into the subdural space. In this case the x-ray views taken from the occipital and posteroanterior directions, disclosed essentially the same findings as seen on pneumoencephalographic pictures and confirmed the fact, that the lesion in this case, was cortical rather than situated in the deeper portions of the cerebrum. In some of our joint publications, we have described this subdural air test and even other modifications of our own. We have however not had the opportunity of stressing its particular value in lesions which are exclusively meningeal or meningocerebral. In this case the lesion is of a leptomeningeal type, ruling out the dura. An ordinary pneumoenceph-

alograph will not serve to make this fine distinction. Prior to our joint experimental and surgical work in epilepsy one of us (Fitch) had extensive experience with many cases in which this method of our study was followed. Under his direction, this method, using two burr holes in most of about fifty patients was followed by another one of us (Weingrow), with the added advantages of the usual pneumoencephalographic tests in the same cases. Our ideas on the mechanics of cerebral circulation, are an outgrowth of the studies of such films, coupled with the clinical and historical data.

In the presentation of the above cases, we have recently emphasized the fact that it is important for us to prove that the seat of the lesion in this syndrome group is located in the cerebrum. We did this in almost every case. In the course of the description of the cases we were aware of the fact that we were piling up a

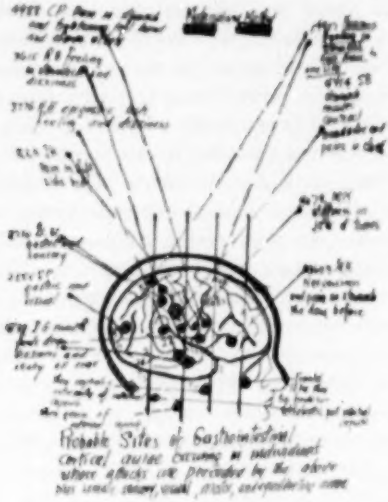

Fig. 74

great deal of clinical material which will need elaboration. We realized that the reader will require explanations of a great many of the facts which we were presenting. An appreciable number of these assembled facts, called for comment which we made strenuous efforts to resist, because we did not feel like disrupting the main purpose of our marshalling of evidence to focus the attention upon localization of the lesion in our cases in the cerebral hemispheres, exclusively. From time to time we were attracted to the fact that disturbances in gastrointestinal function might in themselves lead to metabolic changes which would influence the production of convulsions. It is well known that the rates of absorption of various chemical substances of the food as it passes along the gastrointestinal tract, may be influenced by changes in the gastrointestinal musculature or the stretching or erosions of the mucous membranes. Relaxation of the sphincters of

portions of the gastrointestinal tract leading to the liver, pancreas or other portions, might likewise affect the digestion as well as absorption of the food constituents. Problems such as the above we were repeatedly resisting, since they called for comments on the relationship of our clinical material to the experimental work of various authors. The work of Gibbs, Lennox, and Gibbs (1940) on the variations in the carbon dioxide content of the blood in epileptics seemed relevant to the above. A particularly important paper in the above connection is also the one by Gibbs and Lennox in 1939, dealing with the influence of the blood sugar level on the bioelectric curves.

Along the above lines of investigation of Lennox and his coworkers we had the impulse to elaborate upon the work of Weiss and Fabrykant on the excretion of the bisulfite binding substances in their relation to carbohydrate metabolism. Of particular interest with reference to the same was also the work of Pacella and Fabrykant on acetylcholine in its relation to carbohydrate and sodium metabolism. Although the above subjects are extremely relevant, since they could explain the seizure frequency in some of our epileptic cases on the basis of gastrointestinal motor, secretory, and related upsets we could not allow ourselves to do more than stress their importance with relation to the above as well as their probable bearing on the convulsive mydriasis of Muskens and on the convulsive myosis of Pollock and Furstner. To the last mentioned authors we have amply referred in the first half of this publication.

Another item which we were urged from time to time to comment upon was the therapy in our cases. We merely mentioned in a number of our patients that certain drugs such as phenobarbital were administered. It is not unlikely that other drugs given in combination with the above, such as belladonna, would be effective. Such a combination is favored by Kilstein according to his publication in The Review of Gastroenterology in 1947. We feel that we do not know enough about the present subject to favor the above drug combination or pituitary-like equivalents such as prostigmine, pilocarpine, etc., etc. No one will deny, however, that attempts along either of the above lines must lead to the gateway of those corticodiencephalic paths which lead to the highest autonomic centers in the central nervous system. Another subject which we were repeatedly anxious to elaborate upon in our cases was that of the mental aspects of our patients. In our presentation of the literature we referred now and then to neurosomatic deterioration in epileptics. Here was our chance to dwell further upon a most important subject and to discuss the various types of deterioration. We again felt that due to the urgency of firmly establishing exclusive cerebral control, this was not the place to spend time upon intellectual or emotional deterioration no matter how important such a subject might be.

Aside from the above subjects of metabolism and mental disturbances in the pain syndrome group, the subject of pneumoencephalographic and particularly x-ray findings in the above cases, required some additional comment. For some unknown reason x-ray of the skull has been neglected in medicine as compared with pneumoencephalography. The anthropologist has paid far more attention to

the shape of the skull than has the neurologist. With the exception of the erosive evidences of increased intracranial pressure and fracture lines we know next to nothing about principles of the physics involved in the shaping of the skull. Our work with pneumoencephalcgraphs shows that intracranial pressure is a tremendous factor in brain pathology. It seems not inconceivable to suppose that our knowledge of flight, fatigue, dimout and blackout phenomena, of the mechanics involved in cerebral hemorrhage, and the basic principles involved in ventricular shifting and shape and size changes of the ventricular channels in internal and external hydrocephalus, would be placed on a firmer footing, were we to know more about skull flattenings, skull bulgings and skull size, shape and thickness in relation to other physical characteristics. We felt that the roentgenographic findings in our patients should have been elaborated upon in greater detail than they were. In our first joint publication in 1938 we presented some of these findings in epileptic patients in tabular form. One particular phenomenon seems relevant in our diencephalic cases. This is the anteroposterior contraction at the base of some of the skulls of the epileptic patients. Such a contraction would tend to affect the basal structures. One of us (Pigott) devoted a great deal of his roentgenographic efforts to the above phenomenon. Not only is the above relevant to our cases, but various depressions, thickenings, hyperostoses and flattening of the skull, particularly in the frontal region, seem noteworthy particularly since, the centers of neurogenic function of the gastrointestinal tract are localized therein. The frontal lobe is of special interest in our visceralgic and other material because its frontopontine system passes into the thalamus directly as seen in Bing's diagrammatic construction of the internal capsule constituents. Although we do not wholeheartedly share the above view, no matter how fond we are of the corticocerebellar system we most emphatically agree with the elaborate role played by the frontal lobe in gastrointestinal operations (see Figs. 1-4, 11, 39, 40, 42, 43).

For a number of years our collaborator has consulted the works of various anthropologists in respect to the contraction at the base, and the skull shape of the frontal and other regions. There is no doubt that the progress in the development of the frontal lobe of various races is an important item in the above connection. The assumption of the erect posture in the human, as compared to other forms, depends upon developmental processes such as the above. Skull phylogeny furthermore dates back to great antiquity. We see that in the findings of the Java skull which goes back almost 500,000 years. We also see the progress of the frontal lobe related to that of the higher apes in the above skull type and through that of the Cro-Magnon man, whose existence dates back between 15,000 and 20,000 years (see Figs. 30, 45, 46, 48, 52, 53, 54, 56-66).

From the above everyone can realize how important the frontal lobe really is. Its functions of coordination and equilibration, judgment, feeling tone, as well as gastrointestinal regulation are all indispensable to the understanding of our group syndromes. We feel, however, that the above is not as important as the understanding of the special neurones that extends from the cerebral cortex down to the opposite side of the spinal cord or the constituents of the internuncials and

final common pathway. This cellular unit in the form of a bicellular or tricellular collection really forms the basis of many symptoms as well as clinical neurological findings which we have stressed and will continue to note in our cases. Furthermore, this unit is much older than the progress of the frontal lobe which as we have shown is over 500,000 years in development. The frontopontine pathway has been stressed as having an exclusive connection with the thalamus by some authorities (Bing, 1938). These and similar facts should orient the gastroenterologist and direct him to this portion of the cerebrum as subserving gastrointestinal control and functional integration of viscera more than any other part of the cerebral hemisphere. The bicellular and tricellular collections of fibres constitute the projection systems to the cerebral cortex. All of our past efforts at directing the reader to remember certain outstanding facts seem insignificant and almost unimportant compared with the following. That is, that the data in the preceding

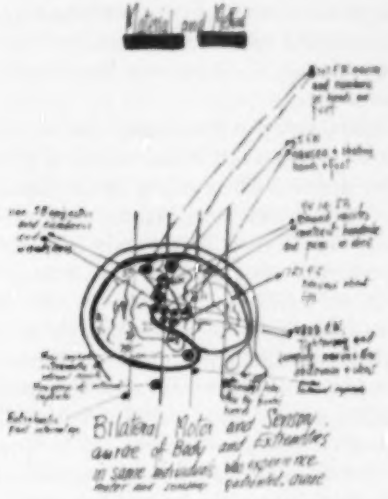

Fig. 75

five paragraphs and especially that of the last few, is intimately bound with the work of Greving on the anterior nucleus of the thalamus, with that of Malone and Morgan on the cellular constituents of structures making up the mammillothalamic pathway and with the efforts of Cajal, d'Hollander, Wilson, Muskens, Le Gros Clark and others whose experimental reports established the fact that the mammillothalamic system is an important part in the mesial and lateral faces of the frontal lobe. It is the projection system which explains some of the symptoms of abdominal pain as well as certain motor disturbances which precede epileptic attacks in the form of aurae. In Figure 74 we see these aurae in the form of pain in the abdomen, visual disturbances as well as vertigo. Other phenomena which we noticed in most of the cases presented above are those not only of the convulsion proper but also those which were especially observed in the course of the neurological examination. These consist mostly of motion, muscle strength, co-

ordination, and trophic disturbances. A definite understandable explanation for the mechanism behind such functions as well as of the convulsion can be seen only in our understanding of the ways in which this bicellular and tricellular projection and association system operates. For some time we have been anxious to stress the simplicity behind the projection and association systems in both cerebral hemispheres. Cajal has done that for us in his schematization in which the long fibre bicellular tracts are combined by collaterals with the corpus callosum above and with the anterior commissure which we neglected anatomically. We do not wish to neglect it clinically. We incorporated it into some of our first ten illustrations. We therefore suggest that the reader repeatedly refer to it and the relation it has to our mammillothalamic pathways. As far as we know, there are no specific syndromes pointing to it but we earnestly feel that the bilateral character behind many of the visceral aurae might be ascribed to the intercerebral commissural connections which this pathway has. In Figure 75 we see the exact location of some of the patterns that are controlled by the projection systems. The intercerebral association systems belong to the corpus callosum, the anterior commissure and similar structures.

Besides our conclusive proof to the reader that in our pain syndrome group we are dealing with cases which suffer from the cerebral lesions, we have also to show that various other diseases which might be accompanied by headache, vomiting and convulsions can be eliminated as not present in the material of the present syndrome group. To do so, it is necessary not only to utilize the result of our findings on a neurological examination but to adduce laboratory data of the blood, urine, x-ray, and of various other tests. In the following way we utilize such tests to eliminate other diseases; ailments such as diabetes, polycythemia, lead encephalopathy, renal disease, uremia, and sinus thrombosis are all ruled out by tests on the blood, urine, as well as by the clinical history. Brain abscess as well as meningitis can also be ruled out by the above methods, including lumbar puncture. There is, however, a group of various other diseases such as multiple sclerosis as well as spinal diseases which require special attention. It is these disease syndromes in which we also find many of the disturbances that are noticed in our pain syndrome group. In these other diseases particularly multiple sclerosis, encephalitis, etc. we also find trophic changes as well as paralyses. The absence of convulsions and gastrointestinal aurae in such disease symptoms are alone insufficient to differentiate these other diseases from our cases. One has to show that the involvement is intracranial and particularly cerebral in our cases but not necessarily in the others. Since those functions that are involved in motion and in muscle strength testing are so important, it is essential that we have at least some idea of the mechanism by which the projection systems operate along motor, reflex, and sensory functions in the form of bicellular and tricellular patterns. Every neurologist knows that without a fundamental knowledge of the structure and function of these long axone pathways, the exact localization of lesions in the above diseases would be impossible. Such knowledge, not only aids in this but seems to simplify the mechanism behind the functioning of alternating

reflex operations which seems so essential in the function of somatic and autonomic body structures. The ideas of bicellular and tricellular somatotopographic arrangement, is the indirect result of the efforts by Cajal and Rosett at neuroanatomic simplification. With such efforts tending toward one goal, it should not be surprising if the anatomic and clinical material should aid in the understanding of the mechanism behind alternating reflexes both somatic and autonomic. In the past we referred in great detail to the work of Davison and Goodhart, to the experiments of Muskens, Pavlov, and finally Penfield. The above authors dealt especially with phenomena which definitely fell into the functions of the projection systems. It is interesting to note that Pavlov, who was essentially a physiologist contributing greatly to the relationship between the functions of the cerebral cortex and the gastrointestinal system, did not furnish us with a good understanding of a more extensive anatomical representation of his investigatory reflex. He stated for instance, that in a decerebrate animal, although this investigatory reflex is intact, yet the animal will die if left alone. Pavlov did not stress the fact that his investigatory reflex is not confined necessarily to the brain stem but extends into the cerebrum. The role played by the investigatory reflex mechanism in salivary and related gastrointestinal functions, has been previously emphasized, and its importance in conditioned reflexes is familiar to every neurophysiologist. We can see how the bicellular system operating even as a reflex unit is important in many ways. R. Lorente de No states that the elementary reflex arc is still an anatomic idealization. He proves, however, by referring to Coghill, that the bicellular system extending longitudinally from the cortex to the metameres of the swimming apparatus, is much older than the reflex arc. There seems to be no doubt that the ultimate decision as to the exact mechanism behind the functions of the cerebral cortex will eventually be determined by the work of the present school of bioelectric neurophysiologists. The work of Lorente de No on internuncials, on nystagmus, on the trigeminal, and labyrinthine reflexes, and particularly on eye movements are all important and especially is the last one mentioned as related to the work of Davison and Goodhart and the experimentalists to whom these authors referred. The tendency throughout our paper, of stressing the bicellular somatotopographic approach, and ascribing the same to an implication of it in a neuroanatomic diagram of Rosett is not just a fancy of ours. It may be related to a tendency at physiologic simplification because of our great respect for the neuroelectric school of physiology. Our tendency in the bicellular and tricellular direction really has a differential diagnostic background. In one of his amazing simplifications of the cerebral projection and association scheme, Cajal hooks up both cerebral hemispheres with each other by the corpus callosum above and by the anterior commissure below. This he did by collaterals and by a long bicellular and tricellular tract association scheme. It is only such an arrangement that can explain the convulsive and gastrointestinal pictures in our cases, and we don't care, if Lorente de No calls them internuncials or Pavlov reflexes. Whatever the mechanism involved in differentiating between reflex action and motion, and the resulting abnormal movements such as tremor, chorea or clonus, we ourselves

cannot afford to indulge in the luxury of speculation, particularly if conclusions drawn, must be with regard to laboratory results. We are for instance, not interested as to whether the gastrointestinal movement reaction to the stimulation of the cerebral cortex, is a reflex response, or because of its continuity, it is a motor response. The above applies also to the mechanism behind prolonged chain reactions seen in conditioned reflexes. We are merely interested in the symptoms as shown in Figures 74 and 75 in relation to their practical application in localizing diagnosis. As such, these symptoms, plus the clinical neurological findings, have enabled us to place the lesions, which the patients that we presented above suffered from, in the cerebrum exclusive of anywhere else in the nervous system. We have prevously enumerated a series of conditions from which one has to differentiate the clinical epileptic visceral pain syndromes, which occurs in our cases. We showed how spinal diseases, polycythemia and other conditions must be segregated from our syndrome group, by laboratory and clinical signs as well as by the special way of taking of a history. Although we have followed a certain etiological classification in our past publications, and in our intramural as well as in the extramural patients, our first concern was to differentiate our convulsive cases, from those, having such diseases, that brain tumor must be eliminated as a possibility, we often felt that such routines are not encompassing enough. The method of differential diagnosis employed by us above is sufficient as a routine procedure in the average collection of cases in which traumatic conditions are the most frequent causes of fits, and infections and degenerative conditions, come next. Other diseases such as alcoholism, heat stroke, congenital defect, familial disease, hypertensive encephalopathy and cerebral arteriosclerosis succeed the former classes of diseases causing fits, but occur less frequently in a collection of patients, than do the former (see Figs. 30, 45, 46, 48, 52, 53, 54, 56, 66).

In his ultimate decision with regard to all possible conditions that might cause the epilepsy and the gastrointestinal syndrome, we suggest that the reader employ a wider range of medical conditions than that found in our publications thus far.

A very satisfactory classification is that offered by A. Subirana. It follows the scheme of Alpers. Under the heading of intoxications he considers botulism, tetanus, submersion, cocaine, lead, epinephrine, arsphenamine, picrotoxine, thujone, caffeine, nicotine, camphor, anesthesia and other intoxicants. Under the caption of general medical conditions he places protein sensitization, hyperventilation, hypertension, subacute bacterial endocarditis, Stokes-Adams-Syndrome, Carotid-Sinus-Syndrome, uremia, hypoglycemia, hypoparathyroidism and blood dyscrasias. Otherwise Subirana's list compares well with ours except that he added such conditions as cerebral aplasia, cerebral hemiatrophy, tuberous sclerosis, lobar sclerosis, presenile dementia, Raynaud's disease, angioneurotic edema, and cerebral vascular spasm. Some of the above were so infrequently encountered by us that we did not consider them worthy of mention while others we placed under general headings such as cerebral arteriosclerosis.

There remains, however, a specific group of diseases such as migraine, Ménière's syndrome, and finally tic douloureux which is not so easy to differentiate

from our cases, except by the absence of convulsions in cases of the former. Pains of all types, scotomata as well as vertigo and vomiting seem to occur with the other conditions as they do in our cases. In these conditions it seems, that besides the absence of convulsions, there is also an absence of extensive positive neurological findings as compared with the cases which we have presented in our pain syndrome group, excepting the Ménière's syndrome. The attacks in these diseases are usually longer lasting, resembling the epileptic prodromes. In dealing with the previous syndrome of visceral pain, we made strenuous efforts to present detailed discussions after every case. We did so because we were anxious to impress the reader with the fact that in all of our syndrome cases, we are dealing with lesions which are essentially cerebral, even though the original cerebral lesion might be instrumental in the production of descending degenerative changes extending into the mesencephalon or lower.

In the following series of cases we present merely the "gastric" syndrome. No matter what descriptive data we offer with this syndrome group we wish to emphasize here and now that the term "gastric" was used merely to cover all types of aurae in cases in which the examiner could not elicit from the patient or his family the exact nature of the gastrointestinal aura. Because of the eventual death of most of the patients, a detailed follow-up was impossible in order to ascertain as to whether the aura was sensory, motor, or even psychic. In spite of the above indefiniteness we will make efforts to qualify these cases by separating them into various categories of "gastric" aura. In Figure 76 we present a view of the left cerebral hemisphere upon which may be seen large black circles which represent the centers of localizations of the various types of the so-called "gastric" aura. The first division of "gastric" aura cases covered about 82 individuals. In 70 of these patients, merely the term "gastric" was used. In the remaining 12 cases, the so-called "gastric" aura, was associated with a peculiar feeling, a sensation referring to the head associated with disturbances in smell, a "gastric" aura associated with visual disturbances and finally a "gastric" aura accompanied by or preceded by a cry. Should we in the future fail to stress the "cry syndrome" of the thalamus while dwelling upon the syndromes related to the anterior nucleus of that structure, let the reader remember that though it is a seemingly minor item it has a tremendous literature. This literature includes such names as Wilson, Beevor, Brissaud, Broadbent, Burzio, Bell, Bard, Goltz, Bechterev, Ferrier, Karplus, Giannuli, Schaefer and others. The average neurologist, merely glancing at these names, will be convinced of the fact that localization of the epileptic cry in the thalamus or in the corticothalamic fibres, is not a matter of theory. When the visceromotor syndrome is presented by us it will be shown that this effective response is only one of its six or seven motor or efferent reflex reactions. Another division of the so-called "gastric" aura syndrome, consists of sensations which are referred to the above portion of the abdomen. In 6 of these cases, merely the term "epigastric" was given. In 2 cases, the "epigastric" aura was combined with dizziness. In 2 other cases, the "epigastric" aura was associated with numbness and weakness. In 2 more cases, the above aura was accompanied by nervousness and distress. In

one case of this group, the so-called "gastric" aura the convulsion was preceded by constipation. In addition to the above 82 cases, there were 20 more individuals in this group whose "gastric" aurae were associated with choking sensations, peculiar sensations about the mouth, peculiar chewing movement, twitching about the mouth, sucking movements and even one case of Jacksonian epilepsy. In spite of the great number of cases in the "gastric" syndrome group as compared with other syndrome groups, it is the least important one, because the term "gastric" does not convey anything of a specific nature such as the term "pain" or "vomiting". For this reason we propose to place the presentation of some of the individual choking sensation cases of this syndrome into the visceromotor syndrome group. We chose to take the choking sensation and the grinding movement aura cases out of this group and are placing these with the visceromotor syndrome. These cases can in

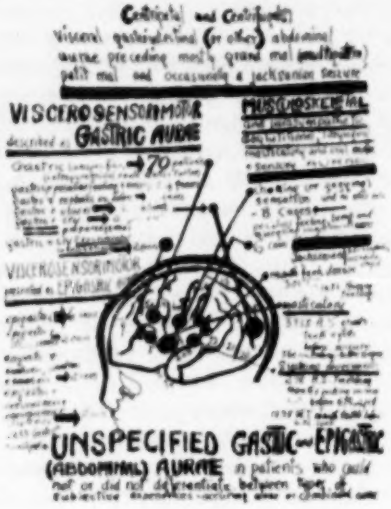

Fig. 76

fact be placed with any of the syndromes since they are not pure gastrointestinal aurae being a mixture of paroxysmal somatic and parasympathetic types of reactions and sensations. An analysis of the disposition incidence of 68 persons, of this group of over 100 cases, will show that few of these patients were available for examination. Most of these individuals were admitted to the institution during the early part of the present century, when special care in questioning patients for specific aurae was not exercised as often as it is today. For this reason, it will be seen, that complete data of a number of patients whose case histories were analyzed, were not available at the time when a neurological examination was meditated. Out of 68 case histories studied in this "gastric" syndrome group, thirty-nine of the patients were dead at the time the study was made. Sixteen of the patients had been discharged at the time of the analysis. One patient in this group, was transferred to another institution. There were only 12 patients out of

the 68 case histories in this group, who were living at the time of the examination, and who could therefore be questioned personally with regard to rechecking aura, and these were studied in relation to the history and the neurological findings. In Figure 76 which we presented with the "gastric" syndrome group, there is noticed some reference to dizziness combined with the other aurae. This type of sensation is not rarely met with, in almost every syndrome group. It is a complicated symptom, to which the patient occasionally refers as vertigo. This symptom is encountered in a number of brain diseases especially in Ménière's syndrome. Although dizziness refers to the vestibular mechanism, and forms an interesting symptom of the diseases of the cerebellar system, we cannot at this time devote too much space to the various fibre tracts that are connected with this symptom complex. We previously alluded to the paper by Lorente de No on the vestibuloocular reflex arc. When we did so, we failed to emphasize the fact that there is a tremendous literature dealing with this system, and especially with Russel's fasciculus or the tractus cerebellovestibularis et spinalis. The vestibulocerebellar mechanism has further been worked-up by Bruce, Klimoff, Edinger, Winkler and others, including Muskens. The above statements apply likewise to the cerebellomotorius fibre system. All the data will be taken into consideration in our dealing with the relationship between the cerebrum and the cerebellum in regard to our neurological findings.

(To be continued)

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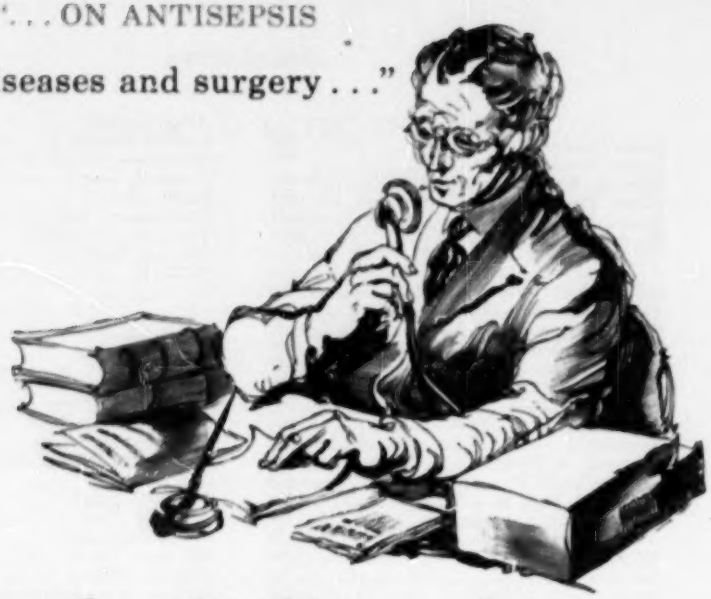

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1. Weiss, S., et al.: Rev. Gastroenterology 16:501-509 (June) 1949.

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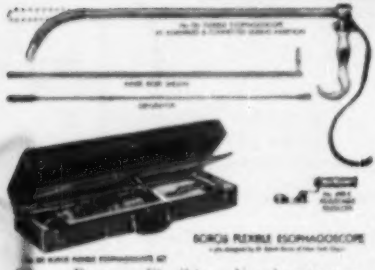

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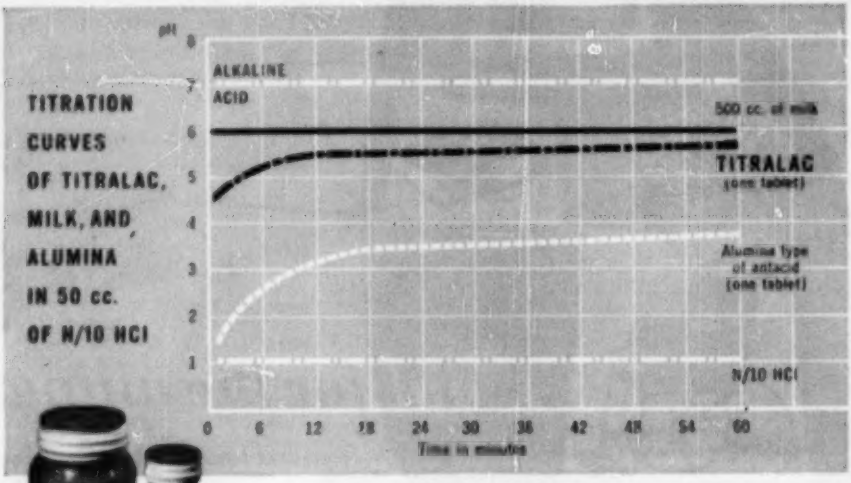

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REFERENCES

1. Rossett, N. E., and Flexner, J.: Ann. Int. Med. 18: 193 (1944). 2. Freezer, C. R. E., Gibson, C. S., and Matthews, E.: Guy's Hosp. Reports 78: 191 (1928). 3. Aaron, A. H., Lipp, W. F., and Milch, E.: J. A. M. A. 139: 514 (Feb. 19) 1949. 4. Kirsner, J. B., and Palmer, W. L.: Illinois M. J. 94: 357 (Dec.) 1948. 5. Kimball, S.: in Practice of Medicine (Tice). Hagerstown, Md., W. F. Prior Company, Inc., 1948, p. 210. 6. Special Article: M. Times 76: 10 (Jan.) 1948.

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2 cakes or packages yeast
¼ cup warm (not hot) water
1 tablespoon salt
1½ tablespoons sugar
2 tablespoons melted vegetable shortening
2¼ cups mashed ripe bananas* (5 to 6 bananas)
5¼ to 6 cups rye flour

**Use fully ripe bananas . . . yellow peel flecked with brown.*

Dissolve yeast in water. Mix together salt, sugar, shortening and bananas. Add half the flour and beat until smooth. Beat in the dissolved yeast. Add remaining flour gradually and mix well.

Turn dough out onto a floured board. Knead about 8 minutes, adding just enough additional rye flour to prevent sticking. Place dough into a lightly greased bowl. Cover and let rise until double in bulk (about 2 hours).

Turn out again onto floured board and knead lightly about 2 minutes. Shape dough into 2 loaves. Place into lightly greased bread pans (8 x 4 x 3 inches). Cover and let rise again until double in bulk (about 1 hour).

Bake in a hot oven (425° F.) 5 to 10 minutes, or until crust begins to brown. Reduce temperature to 350° F. and bake 35 to 40 minutes longer, or until bread is done. Remove from pans. Brush top crusts with water. Makes 2 loaves.

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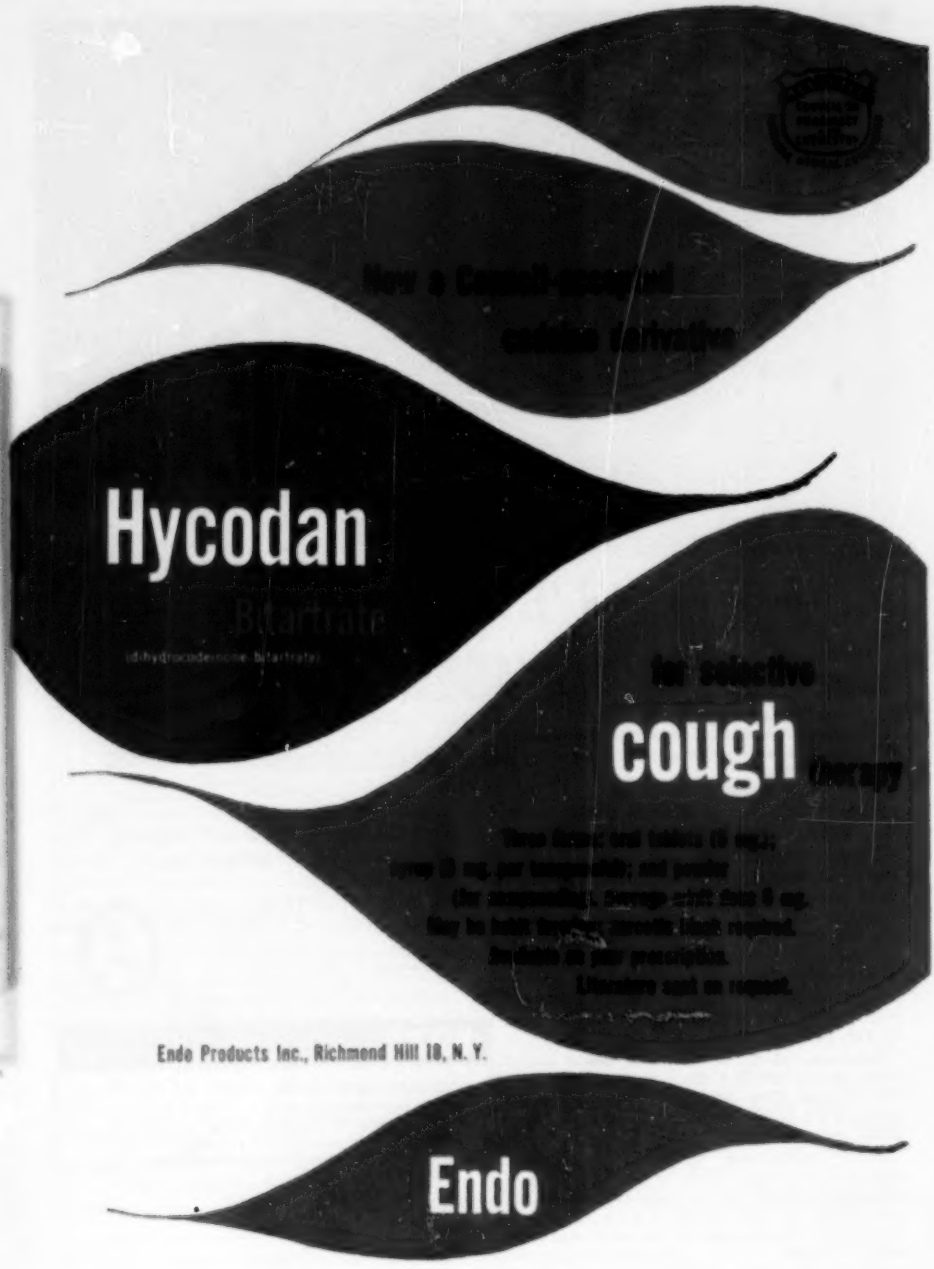
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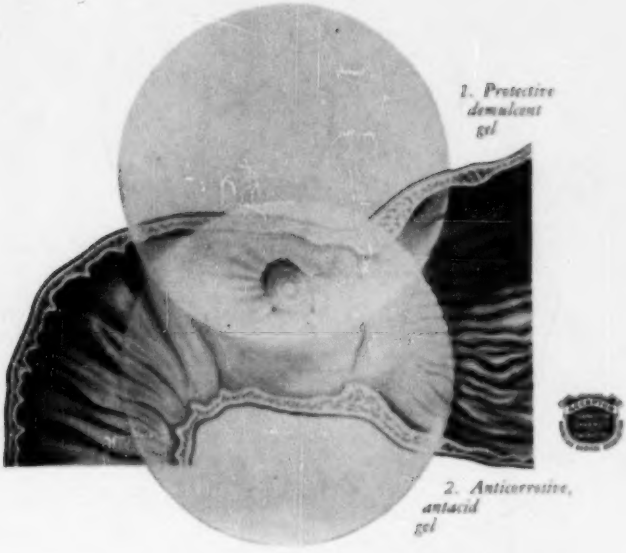

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